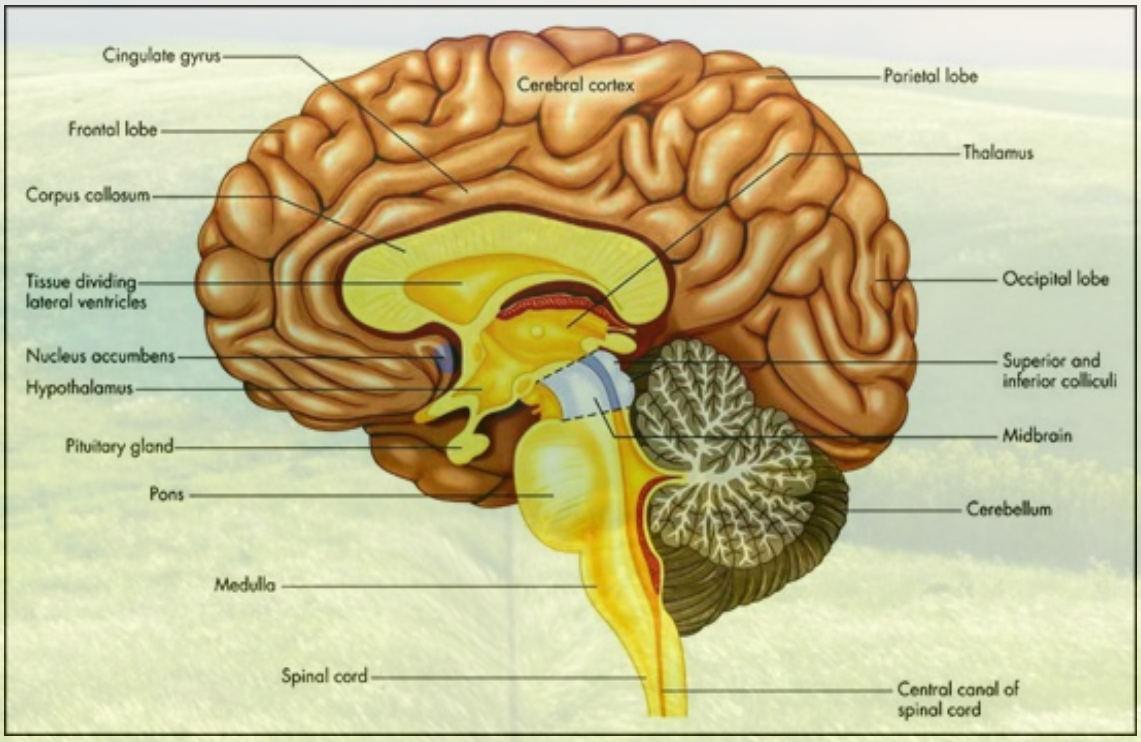
NEUROLOGY



Sherif EL Hawary, MD

INDEX

CENTRAL NERVOUS SYSTEM

The CNS is formed of 2 parts:

I. INTRACRANIAL PART

1. Two cerebral hemispheres.
2. Basal ganglia.
3. Brain stem.
4. Cerebellum.

1. Two cerebral hemispheres:

- CONNECTION: They are connected to each other by the CORPUS CALLOSUM.
- SURFACE: The surface of each hemisphere is divided into 4 LOBES:
  1. Frontal. 2. Parietal. 3. Temporal. 4. Occipital.
- CEREBRAL LOBES:
  1. *Outer gray matter:* *(cerebral cortex)*
     - It is composed of: NERVE CELLS.
     - It contains certain AREAS that control specific functions.
  2. *Inner white matter:* *(depth of the cerebral hemisphere)*
     - It is composed of: NERVE FIBRES.
     - It CONDUCTS IMPULSES to & from the cerebral cortex.

2. Basal nuclei:

- **At the base of each cerebral hemisphere** ***(At various levels deep in the white matter)*****:**
  - BASAL GANGLIA: *Caudate, Putamen, Globus pallidus.*
  - THALAMUS, SUBTHALAMUS, HYPOTHALAMUS.

3. Brain stem: " 3 parts from above downwards "

- MIDBRAIN: contains the **motor nuclei** of the cranial nerves 3 & 4.
- PONS: contains the **motor nuclei** of the cranial nerves 5, 6, 7.
- MEDULLA: contains the **motor nuclei** of the cranial nerves 9, 10, 11, 12.

- The 1st, 2nd & 8th cranial nerves have **no motor nuclei.**
- They are SENSORY NERVES concerned with special sensations.

4. Cerebellum:

- It lies at the BACK & the BOTTOM of the cranium behind the brain stem, in the posterior cranial fossa.

II. SPINAL PART

1. Spinal Cord.
2. Cauda Equina.

1. Spinal Cord:

- It lies in the spinal canal.
- It ends at the lower border of the **1st lumbar vertebra** (L1).

Segments of the spinal cord:

- *Cervical segments:* *8 segments.*
- *Thoracic segments:* *12 segments.*
- *Lumbar segments:* *5 segments.*
- *Sacral segments:* *5 segments.*

- CONUS MEDULLARIS: The lowermost 3 segments of the spinal cord ($S_{3,4,5}$).
- EPICONUS: The 4 segments above the conus medullaris ($L_{4,5}$, $S_{1,2}$).

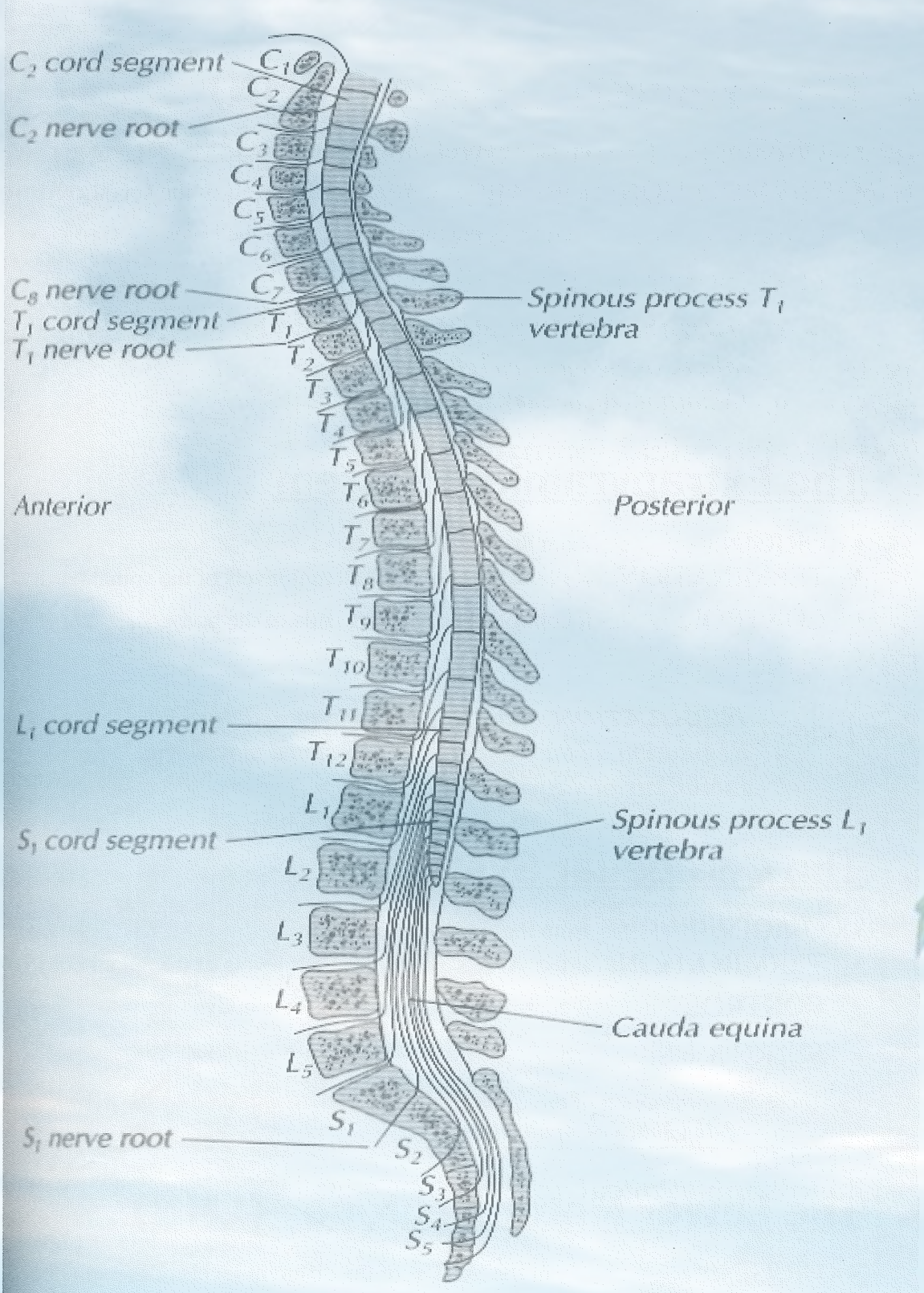
Section in the spinal cord:

- It contains: **gray matter** (cells) surrounded by **white matter** (fibres).
- The **GRAY MATTER**: H – shaped in a transverse section & is formed of:
  - 2 anterior horns: MOTOR function.
  - 2 posterior horns: SENSORY function.
- The **WHITE MATTER**: contains nerve fibres arranged into tracts:

1. *The important ascending tracts are:*
   - Lateral & ventral spinothalamic: for superficial sensations.
   - Posterior column: for deep sensations.
   - Spinocerebellar: for cerebellar information.
2. *The important descending tracts are:*
   - The pyramidal tract (corticospinal tract).
   - The extrapyramidal tracts.
   - The cerebello-spinal tracts.

2. Cauda Equina: "Collection of Lumbo – sacral roots"

- It fills the lower part of the spinal canal.
- It starts at the lower border of the **1st lumbar vertebra** (L1).



THE SPINAL CORD, LATERAL VIEW

THE MOTOR SYSTEM

1. The Pyramidal System (UMN)

- ORIGIN: in the **cerebral cortex** (motor area " 4 " & premotor area " 6 ").
- TERMINATION: at the **AHCs** of the different levels of the spinal cord.
- CONTROL: it controls the **opposite** side of the body.
- FUNCTIONS:
  - ***INITIATION*** *of the voluntary motor activity.*
  - *Inhibition of the deep reflexes.*
  - *Inhibition of the muscle tone.*

2. The Extrapyramidal System

- ORIGIN: from the **basal ganglia.**
- TERMINATION: at the **AHCs** of the different levels of the spinal cord.
- CONTROL: it controls the **opposite** side of the body.
- FUNCTIONS:
  - ***REGULATION*** *of the voluntary motor activity.*
  - *Regulation of the emotional & associated movements.*
  - *Inhibition of the muscle tone.*

3. The Cerebellar System

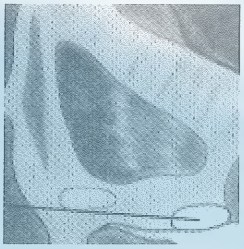
- ORIGIN: from the **cerebellum.**
- TERMINATION: at the **AHCs** of the different levels of the spinal cord.
- CONTROL: it controls the **same** side of the body.
- FUNCTIONS:
  - *Co-ordination of the voluntary motor activity initiated by the pyramidal system.*
  - *Maintenance of equilibrium.*

4. The Lower Motor Neurone (LMN)

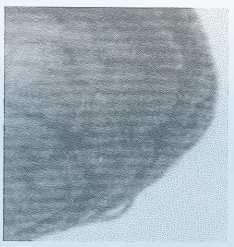
- ORIGIN: in the **AHCs** of the different levels of the spinal cord.
- TERMINATION: at the **voluntary muscles**.
- COMPONENTS: AHCs, PN, NMJ & Voluntary muscles.
- FUNCTIONS:
  - *Transmission of the motor impulse from the **AHCs** to the **voluntary muscles**.*

THE MOTOR SYSTEM

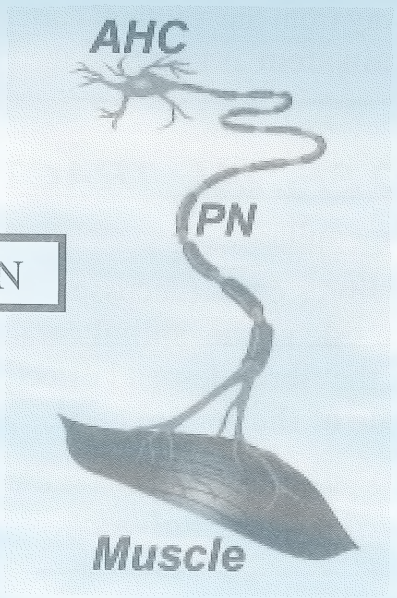
The Pyramidal System
"Controls **opposite** side"



The Extra Pyramidal System
"Controls **opposite** side"



The Cerebellar System
"Controls **same** side"



MOTOR PATHWAY

- ORIGIN: the order starts in the **Cerebral cortex**.
- TERMINATION: on the **Voluntary muscle** which will respond by movement.

From the origin to the termination , the impulse passes through 2 neurones:

1. Upper Motor Neurone (UMN): PYRAMIDAL TRACT.
2. Lower Motor Neurone (LMN): AHCS, Nerves, NMJ, Muscles.

1. UPPER MOTOR NEURONE (UMN)

1. MOTOR AREA " 4 " " CEREBRAL CORTEX "

The voluntary motor impulse originates mainly in the large pyramidal **cells** (Betz cells) of the motor area " 4 " & to a lesser extent in the cells of the premotor area " 6 ".

2. INTERNAL CAPSULE

The **axons** of these cells (carrying the impulse) descend in the depth of the Cerebral hemisphere in the Corona radiata to pass in the Internal capsule, *and*, continue their descent in the brain stem (midbrain, pons & medulla).

3. CORTICOBULBAR TRACT " BRAIN STEM"

In the **Brain Stem**, some of the descending fibres separate to supply the **motor nuclei** of the cranial nerves of BOTH sides except the lower ½ of the facial nucleus and all the hypoglossal nucleus which are supplied only from the opposite pyramidal tract.

These fibres are known as the ***corticobulbar tract*** as they do not reach the spinal cord.

> The pathway from the cerebral cortex down to the **cranial nuclei** in the **brain stem** is known as the " **Corticobulbar Tract** ".

4. CORTICO SPINAL TRACT " SPINAL CORD"

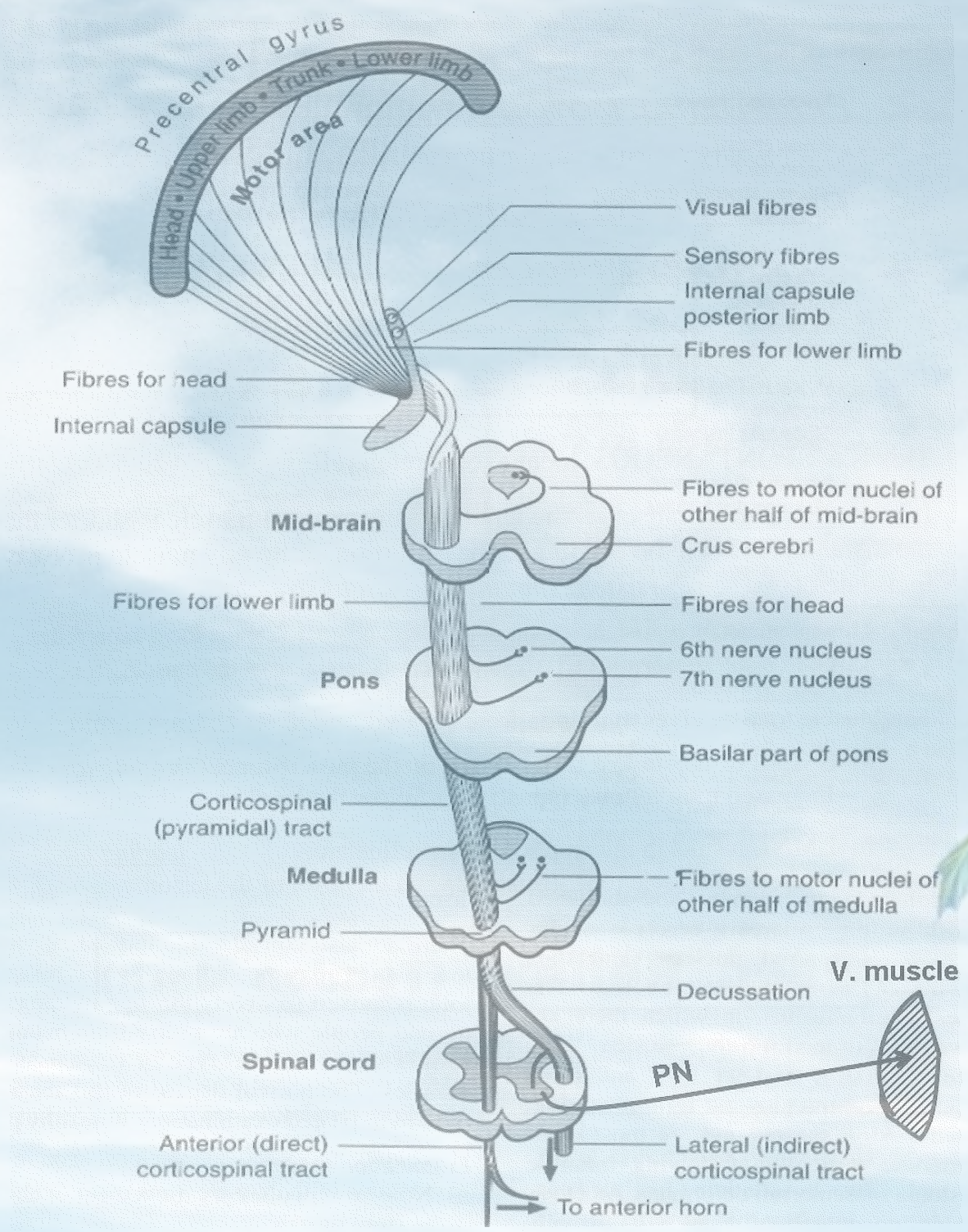
In the lower medulla:

- 85 % of fibres cross (decussate) to descend in the white matter of the **opposite side of the spinal cord**,
- 15 % of the fibres descend directly in the white matter of the **same side of the spinal cord**.

The fibres of the pyramidal tract terminate at different levels of the **AHCs** of the spinal cord ***(corticospinal tract)***.

> The pathway from the cerebral cortex down to the **AHCs** in the **spinal cord** is known as the " **Corticospinal tract** ".

Pathway of the voluntary motor impulse (UMN & LMN)



2. LOWER MOTOR NEURONE (LMN)

1. AHCs

- They are: special type of nerve cells situated in the anterior horns of the: *"H-shaped gray matter of the spinal cord"*.
- They receive the voluntary motor impulse from the corticospinal pyramidal tract.
- Their axons exit from the spinal cord as the anterior roots.

2. PERIPHERAL MOTOR NERVES

- They carry the motor impulse from the AHCs to the voluntary muscles.

3. NEUROMUSCULAR JUNCTION.

4. VOLUNTARY MUSCLES.

Muscle Tone

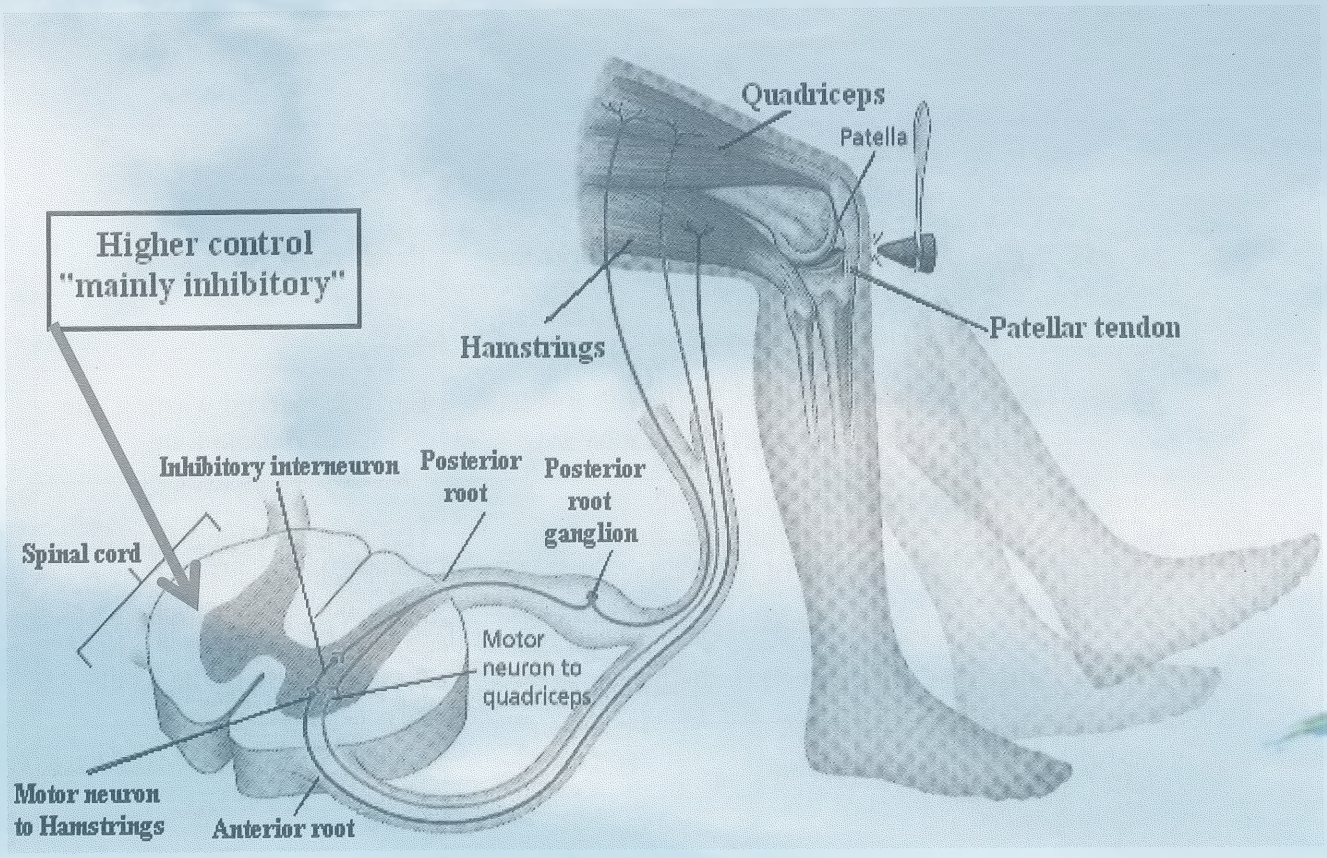
1. This is a SPONTANEOUS local axon stretch reflex.
2. It is spontaneous because: the length of any skeletal muscle is shorter than the distance between the origin and the insertion. So any muscle is always in a state of: ***Persistent spontaneous slight stretch***.
   - This spontaneous persistent stretch will persistently activate the local axon reflex.
   - This will result in **persistent** (maintained) contraction of the muscle (tone).
3. The Muscle tone receives inhibition from the Pyramidal & Extrapyramidal systems:
   a) **UMNL** results in loss of inhibition on the muscle tone, *leading to:* Increased muscle tone **(spasticity)** below the level of the lesion.
   b) **LMNL** results in interruption of the reflex arc, *leading to:* Decreased muscle tone **(flaccidity)** at the level of the lesion.

Deep Reflex (Tendon Jerk)

1. This is an INDUCED local axon stretch reflex.
2. It is induced by: sudden stretch of the muscle by tapping the tendon with a hammer.
   - This induced sudden stretch will suddenly & temporarily activate the local axon reflex.
   - This will result in **sudden transient** contraction of the muscle (jerk).
3. The deep reflex receives inhibition from the Pyramidal system:
   a) **UMNL** results in loss of inhibition on the deep reflex, *leading to:* Increased deep reflex **(hyperreflexia)** below the level of the lesion.
   b) **LMNL** results in interruption of the reflex arc, *leading to:* Decreased deep reflex **(hyporeflexia)** at the level of the lesion.

STRETCH REFLEX

(Muscle tone & Deep reflex)



Clonus

- Sudden sustained stretch of the muscle tendon, results in: *Rapid*, *Rhythmic*, *Regular* contractions.

1. It indicates: Severe pyramidal lesion due to: loss of inhibition on the stretch reflex.
2. It is elicited in the: ankle, patella, wrist.
3. It stops when: the stretch is stopped.

How would you clinically differentiate between:

UMNL & LMNL ??

	UMNL	LMNL
1. Muscle power	Paralysis or weakness below the level of the lesion.	Paralysis or weakness at the level of the lesion.
2. Muscle wasting	**No** wasting, & if present it is late and due to disuse atrophy.	Early & marked wasting due to loss of muscle tone.
3. Muscle tone	**Hypertonia (spasticity) below** the level of the lesion.	**Hypotonia (flaccidity) at** the level of the lesion.
4. Deep reflexes	**Hyperreflexia below** the level.	**Hyporeflexia at** the level.
5. Pathological deep reflexes	May be present.	Absent.
6. Clonus	May be present.	Absent.
7. Superficial reflexes	Lost if the lesion is above the segmental supply of the reflex.	Lost if the lesion involves the supply of the reflex.
8. Plantar reflex (Babinski) *	Positive, i.e. dorsiflexion of the big toe ± fanning of the other toes.	Plantar flexion of the toes, or no response.
9. Fasciculations	Absent	May be present in irritative lesion of the *AHCs*.

* **Joseph Babinski** (1857-1932) was a French neurologist of Polish origin, he described the plantar reflex.

THE SENSORY SYSTEM

- There are **3 types of sensations** in the body:

I. SOMATIC SENSATIONS:

* They are conducted to the CNS via the "**somatic nerves.**"
* They include:

1. *Superficial sensations:*

- Pain.
- Temperature.
- Touch.

2. *Deep sensations (proprioceptive):*

- Vibration sense.
- Joint sense.
- Muscle sense.
- Nerve sense.

3. *Cortical sensations:*

- Tactile localization.
- Two points discrimination.
- Stereognosis.
- Graphosthesia.

II. VISCERAL SENSATIONS:

* They are conducted to the CNS via the "**autonomic nerves.**"
* They include all sensations coming from the internal viscera,
  e.g. *heart & intestines.*

III. SPECIAL SENSATIONS:

* They are conducted to the CNS via the "**cranial nerves.**"
* They include: *Smell, Vision, Hearing.*

Somatic Sensations

- *All somatic sensations, whether superficial or deep pass through* ***3 ORDER NEURONES*** *from receptors in the skin & deep structures to reach the cortical sensory area of the opposite side.*

1. The cell of 1st order neurone is: always in the *posterior root ganglion of the same side*.
2. The cell of 3rd order neurone is: always in the *thalamus of the opposite side*.
3. The cell of 2nd order neurone: varies according to the type of sensation.

A) Pathway of Superficial Sensations

I. Pathway of Pain & Temperature:

1. The 1st order neurone:

- The 1st order neurone is the cell of the **posterior root ganglion (PRG)**.
- The afferent sensory nerve relays in the PRG.
- The efferent sensory nerve enters the spinal cord in Lissauer's tract to relay in the cells of the Substantia Gelatinosa of Rolandi **(SGR)**, at the posterior horn of the gray matter.

2. The 2nd order neurone:

- The 2nd order neurone is the cell of **SGR** & its axon.
- This axon crosses to the **OPPOSITE** side & ascends in the: **Lateral Spinothalamic** tract of the spinal cord, then in the: **Lateral Lemniscus** of the brain stem, to relay in the thalamus.

3. The 3rd order neurone:

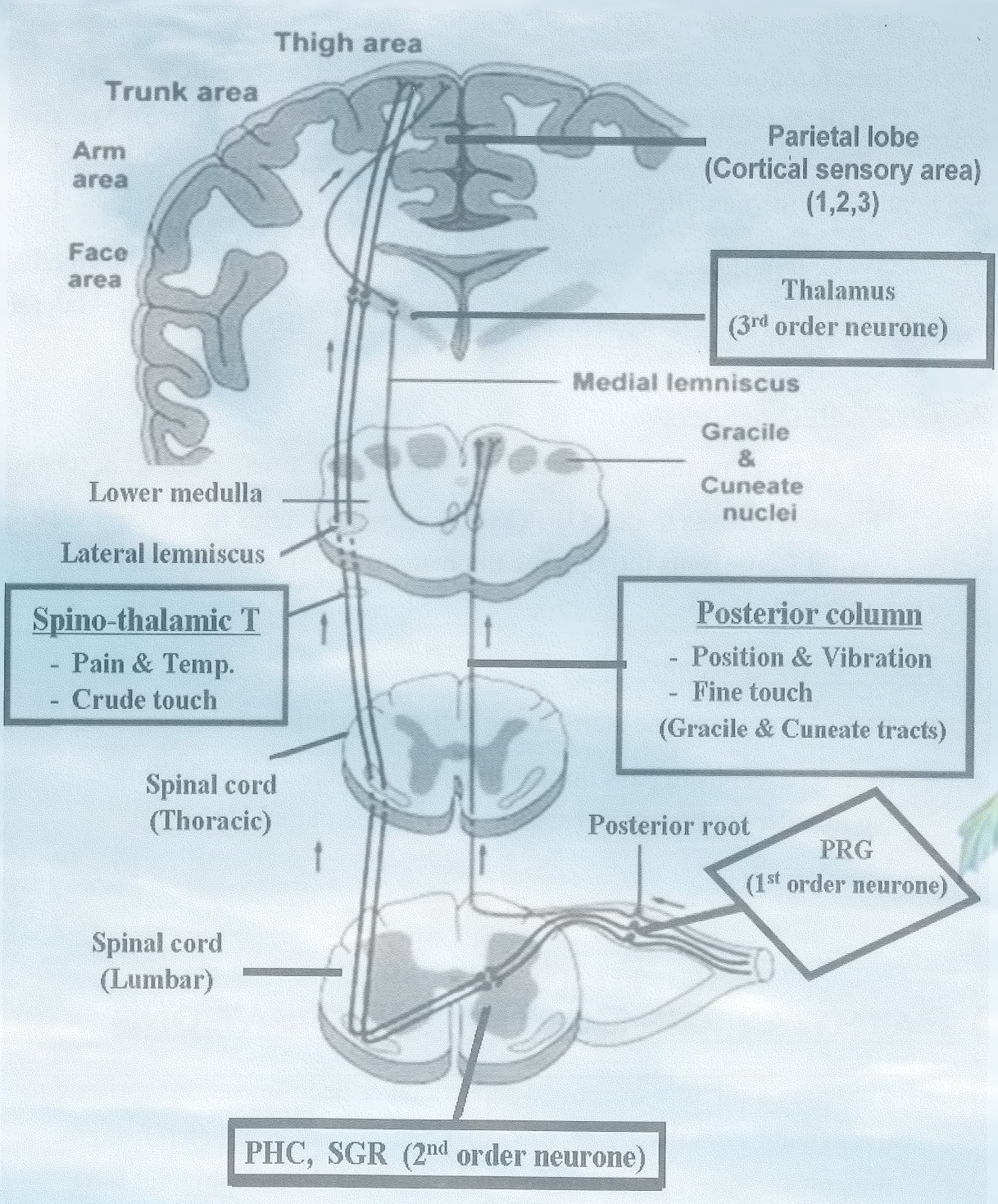
- The 3rd order neurone starts in the cell of the **thalamus**.
- Its axon ascends to pass through the internal capsule conducting the impulse to the cortical sensory area in the parietal lobe.

II. Pathway of Touch:

1. Crude touch: has the same pathway as pain & temperature, BUT: In the 2nd order neurone: it ascends in the **Ventral Spinothalamic** tract of the **OPPOSITE** side of the spinal cord.

2. Fine touch: has the same pathway as deep sensations.

Pathway of Superficial & Deep Sensations



B) Pathway of Deep Sensations

1. ***The 1st order neurone:***
   - The 1st order neurone is the cell of the **posterior root ganglion (PRG).**
   - The afferent sensory nerve relays in the PRG.
   - The efferent sensory nerve enters the spinal cord & ascends in **Gracile & Cuneate** tracts within the posterior column of the **SAME** side (along with fibres carrying fine touch) to relay in Gracile & Cuneate nuclei in the medulla:
     - Gracile Tract: carries fibres from lower ½ of body & lies medially.
     - Cuneate Tract: carries fibres from upper ½ of body & lies laterally.

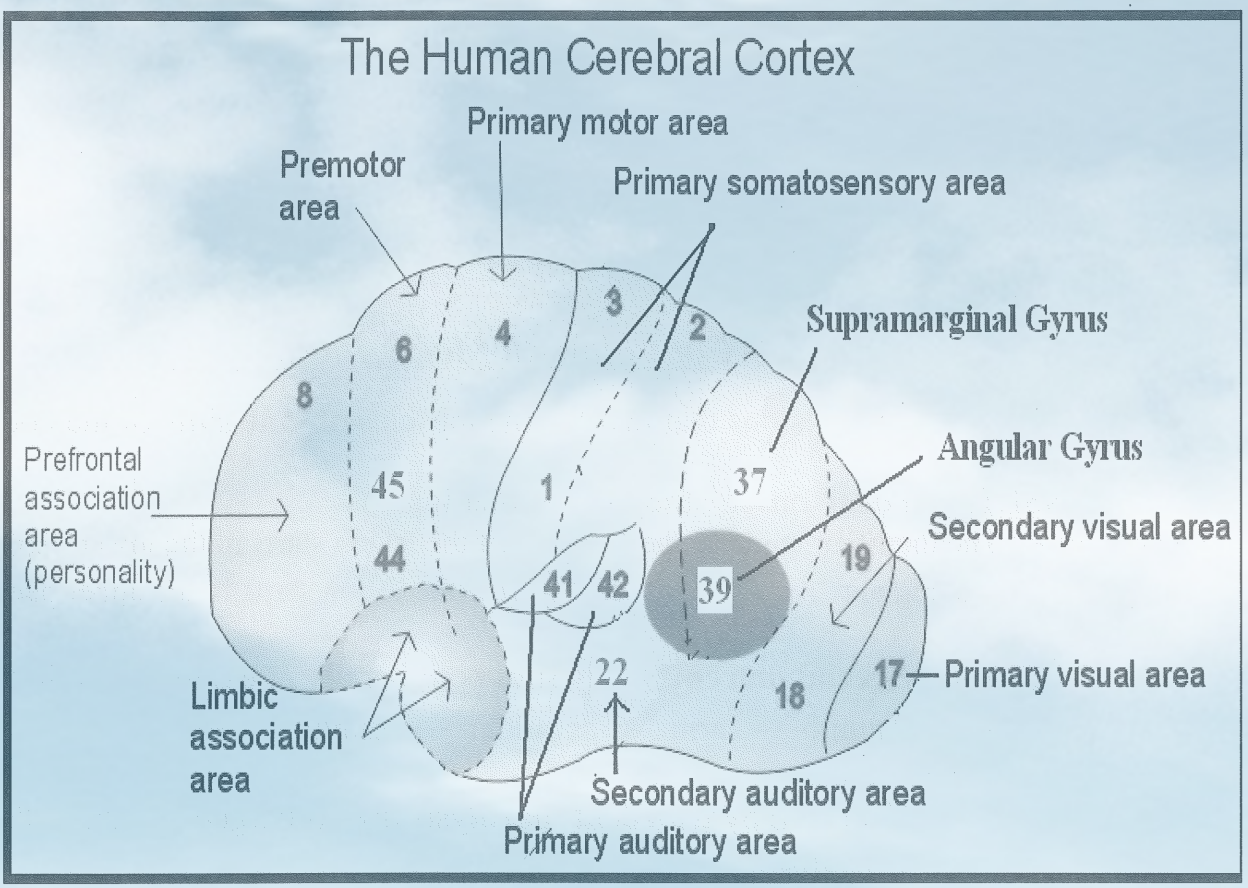
2. ***The 2nd order neurone:***
   - The 2nd order neurone is the cell of **Gracile & Cuneate nuclei** & its axon.
   - This axon crosses to the **OPPOSITE** side & ascends in the **Medial Lemniscus** through the brain stem, to relay in the thalamus.

3. ***The 3rd order neurone:***
   - The 3rd order neurone starts in the cell of the **thalamus**.
   - Its axon ascends to pass through the internal capsule conducting the impulse to the cortical sensory area in the parietal lobe.

C) Pathway of Cortical Sensations

- These are a mixture of refined superficial & deep sensations arriving to the thalamus via the 1st & 2nd order neurones & conducted from the thalamus to the cortical sensory area (1, 2, 3) in the **parietal lobe**.

Areas of the Cerebral Cortex



The surface of each cerebral hemisphere is divided into 4 LOBES:

1. Frontal. *2. Parietal.* *3. Temporal.* *4. Occipital.*

Each lobe consists of: MANY AREAS.

Each area is responsible for a specific function.

FRONTAL LOBE

1. Motor Area (area 4)

- **Function:**
  - Initiation of the voluntary motor activity of the OPPOSITE half of the body.
  - In this area the body is represented upside down.

- **Lesion:**
  - *Irritative:* " *Contralateral Motor Jacksonian Fits* "
    1. SITE: Convulsions that involve the muscles of **one side** of the body.
    2. ONSET: **Focal** onset, they start either in:
       - The thumb or angle of the mouth (if the lesion starts in the lower part).
       - The big toe (if the lesion starts in the upper part).
    3. SPREAD: **Special march:** they start peripherally & extend centrally:
       - **e.g.** Thumb → arm → shoulder → trunk → LL.
    4. OFFSET: Followed by **transient paralysis** due to fatigue of Betz cells:
       - Todd's paralysis.
  - *Destructive:* " *Contralateral Motor Paralysis* "

2. Premotor Area (area 6)

- **Function:**
  - Partly supplies the pyramidal tract.
  - Partly supplies the extra-pyramidal tract.

- **Lesion:**
  - Contralateral Hypertonia & Hyperreflexia.

3. Area of Conjugate Eye Deviation (area 8)

- **Function:**
  - Voluntary conjugate eye deviation to the OPPOSITE side.
- **Lesion:**
  - *Irritative:*
    Attacks of conjugate eye deviation to the OPPOSITE side of the lesion.
  - *Destructive:*
    Loss of conjugate eye deviation to the OPPOSITE side of the lesion.

4. Broca's Area (44)

"*In The Dominant Hemisphere Only*"

- **Function:**
  - It is the Motor center for speech.
  - It is responsible for formulation of spoken words.
  - It is present in the left cerebral cortex in right-handed persons & vice versa.
- **Lesion:** " *Motor Aphasia (Verbal Aphasia)* "
  - The patient cannot express himself in spoken words.

5. Exner's Area (45)

"*In The Dominant Hemisphere Only*"

- **Function:**
  - It is the motor center for writing.
  - It is responsible for formulation of written words.
  - It is present in the left cerebral cortex in right-handed persons, and vice versa.
- **Lesion:** " *Agraphia (Writing Aphasia)* "
  - The patient cannot express himself in written words.

6. Paracentral Lobule

- **Function:**
  - Cortical inhibition (control) of bladder voiding & bowel evacuation.
- **Lesion:**
  - Incontinence of urine & stools.

7. Prefrontal Area

- **Function:**
  - It is the higher center for:
    - Mentality (Memory & Intelligence).
    - Personality (Behaviour, Planning, Problem – solving).
  - It inhibits the primitive reflexes present in the newborn, e.g. *grasp reflex*.
- **Lesion:**
  - Disturbances in: Mentality & Personality → DEMENTIA.
  - Reappearance of the primitive reflexes, e.g. *grasp reflex*.

PARIETAL LOBE

1. Cortical Sensory Area (1, 2, 3)

- **Function:**
  - Perception of cortical sensations from the OPPOSITE half of the body.
  - In this area the body is represented upside down.
- **Lesion:**
  - *Irritative:* "*Contralateral Sensory Jacksonian Fits*"
    1. SITE: Sensory fits (numbness or tingling) in **one side** of the body.
    2. ONSET: **Focal** onset.
    3. SPREAD: **Special march.**
  - *Destructive:* "*Contralateral Cortical Sensory Loss*"

2. Angular Gyrus (39) "*In The Dominant Hemisphere Only*"

- **Function:**
  - It is responsible for **R**eading, i.e. **R**ecognition & **R**ecall of letters & numbers.
- **Lesion:** "*Alexia (Visual Aphasia, Word Blindness)*"
  - The patient is able to see the letters & numbers, BUT,
    He is not able to recognize (understand) them, THEREFORE,
    He is not able to read.

3. SUPRAMARGINAL GYRUS (37) "*In The Dominant Hemisphere Only*"

- **Function:**
  - It is responsible for: Storage & Recall of:
    1. IDEAS of speech.
    2. IDEAS of complex voluntary motor activity.
- **Lesion:**
    1. Jargon's Aphasia (Word salad).
    2. Apraxia: inability to perform complex voluntary motor activity, in absence of: *paralysis*, *sensory loss* or *incoordination*.

TEMPORAL LOBE

1. PRIMARY AUDITORY AREA (41, 42)

- **Function:**
  - It is the center for HEARING.
- **Lesion:**
  - *Irritative:*
    - Auditory Hallucinations.
  - *Destructive:*
    - Slight impairment of HEARING.
    - Never complete deafness because hearing is bilaterally represented.

2. SECONDARY AUDITORY AREA (22) "*In The Dominant Hemisphere Only*"

- **Function:**
  - **R**ecognition & **R**ecall of sounds & heard words.
- **Lesion:** "*Auditory Agnosia*"
  - The patient is able to hear the sounds, BUT, He is not able to recognize (understand) them.

3. Limbic System (Uncus)

- **Function:**
  - It is the center for SMELL.
- **Lesion:**
  - *Irritative:*
    - Olfactory Hallucinations.
  - *Destructive:*
    - Slight impairment of SMELL.
    - Never complete anosmia because smell is bilaterally represented.

OCCIPITAL LOBE

1. Primary Visual Area (17)

- **Function:**
  - It is the center for VISION.
- **Lesion:**
  - *Irritative:*
    - Visual Hallucinations.
  - *Destructive:*
    - Contralateral Homonymous Hemianopia *(bilateral half blindness).*
    - Never bilateral complete blindness because vision is bilaterally represented.

2. Secondary Visual Area (18, 19) *"In The Dominant Hemisphere Only"*

- **Function:**
  - **R**ecognition & **R**ecall of images.
- **Lesion:** " *Visual Agnosia* "
  - The patient is able to see the images, BUT,
    He is not able to recognize (understand) them.

SPEECH DISORDERS

- Normal speech involves 2 stages:

1. Formulation whether Spoken or Written, ... This needs:

A) **Sensory System:**

1. Visual areas: 17, **18**, **19**, and **39**.
2. Auditory areas: 41, 42 and **22**.

B) **Motor System:**

1. Broca's area: **44**.
2. Exner's area: **45**.

C) **Associative System:**

- Supramarginal gyrus: **37**.

Areas 17, 41, 42 are present in BOTH hemispheres.
Areas **18**, **19**, **39**, **22**, **44**, **45**, **37** are present in dominant hemisphere ONLY.

2. Articulation ..... This needs:

A) UMN: Pyramidal tracts.

B) LMN:

- *Cranial Nuclei* (5, 7, 10, 12).
- *Nerves*.
- *NMJ*.
- *Muscles*.

C) Cerebellum: for co-ordination of the muscles of speech.

D) Extrapyramidal system: for making the speech expressive.

- **Disorders of speech include:**

APHASIA

Definition

- Difficulty or inability of the formulation of speech:
  - In the absence of lesions of the sense organs (e.g. Vision or Hearing).
  - In the absence of mental defect.

Types

1. **Sensory Aphasia:** " *due to defect in comprehension* "

   a) Visual:

   - Visual agnosia: (lesion in area 18, 19)

     The patient sees objects but does not recognize them.

   - Alexia: (lesion in area 39)

     The patient cannot read because he does not understand the letters & numbers (word blindness).

   b) Auditory:

   - Auditory agnosia (lesion in area 22)

     The patient hears sounds but does not understand them.

2. **Motor Aphasia:** " *due to defect in expression* "

   a) Verbal aphasia: (lesion in Broca's area)

   The patient cannot express himself in spoken words; (although he can understand visual & auditory stimuli).

   b) Agraphia: (lesion in Exner's area)

   The patient cannot express himself in written words (although he can understand letters & numbers).

3. **Jargon's Aphasia (Word salad):** " *due to defect in association* "

   - It is due to lesion in the supramarginal gyrus (area 37).
   - The patient can speak, **BUT**, the words are meaningless & not related.
   - This condition is usually associated with apraxia.

DYSARTHRIA

Definition

- Difficulty in articulation of speech (with normal formulation of speech).

Types

1. **Slurred Speech:**
   - Disturbance in the production of consonants especially:
     - Labials (e.g. B, M).
     - Dentals (e.g. D, T).
   - Causes include:
     a) *Bilateral* ***pyramidal tract lesion*** *(Cortico-bulbar lesion), as in :*
     - Pseudobulbar palsy.

     b) *LMNL of the cranial nerves concerned with speech, e.g.:*
     - ***Nuclear:*** True bulbar palsy.
     - ***Nerve:*** Facial nerve palsy.
     - ***NMJ:*** Myasthenia.
     - ***Muscle:*** Myopathy (Facio-scapulo-humeral myopathy).

2. **Staccato Speech:**
   - The speech is explosive with separation of the syllables.
   - Causes include: ***Cerebellar lesions:***
     - Hereditary ataxias.
     - DS.

3. **Scanning Speech:** **(Slurred Staccato)**
   - It occurs also in: Cerebellar lesions.

4. **Monotonus Speech:**
   - The speech is expressionless & monotonus.
   - Causes include: ***Extrapyramidal lesions:***
     - Parkinsonism.

THE CRANIAL NERVES

1. OLFACTORY NERVE

- **Function**
  - Sense of smell.

- **Lesion**

1. **Anosmia:** *" loss of sense of smell "*

   a) Bilateral anosmia: causes:
   - ENT causes, e.g. atrophic rhinitis.
   - Congenital.

   b) Unilateral anosmia: causes:
   - Traumatic: fracture cribriform plate (in fracture base of skull).
   - Inflammatory: basal meningitis.
   - Neoplastic: meningioma of the olfactory groove.

2. **Olfactory Hallucinations:** *" false perception of bad smell "*
   - It occurs in *irritative* lesions of the limbic system in the temporal lobe.

2. OPTIC NERVE

- **Pathway**
  - From the retina, the nerve fibres converge to form the **optic nerve**.
  - The ***nasal fibres*** of the nerve (which carry the temporal field of vision), decussate at the **optic chiasma** to pass in the optic tract of the ***opposite side***.
  - The temporal fibres of the nerve (which carry the nasal field of vision), pass in the **optic tract** of the same side.
  - The fibres relay in the lateral geniculate body, where new fibres arise & pass in the posterior limb of the **internal capsule**.
  - The fibres then pass in the **optic radiation** (in the parietal & temporal lobes).
  - The fibres of the optic radiation finally terminate in **area 17** of the **occipital lobe** (visual sensory area), where vision is perceived.
  - The neighbouring **areas 18 and 19** (visual associative areas) are responsible for the recognition & recall of images.

- **Function**
  - Sense of vision.

- **Lesion** differs according to the site in the pathway:

1. **Lesion in the Optic Nerve:**
   - Ipsilateral loss of vision.
   - Loss of direct & consensual light reflex.

2. **Lesion in the Optic Chiasma:**
   - Bitemporal Hemianopia.

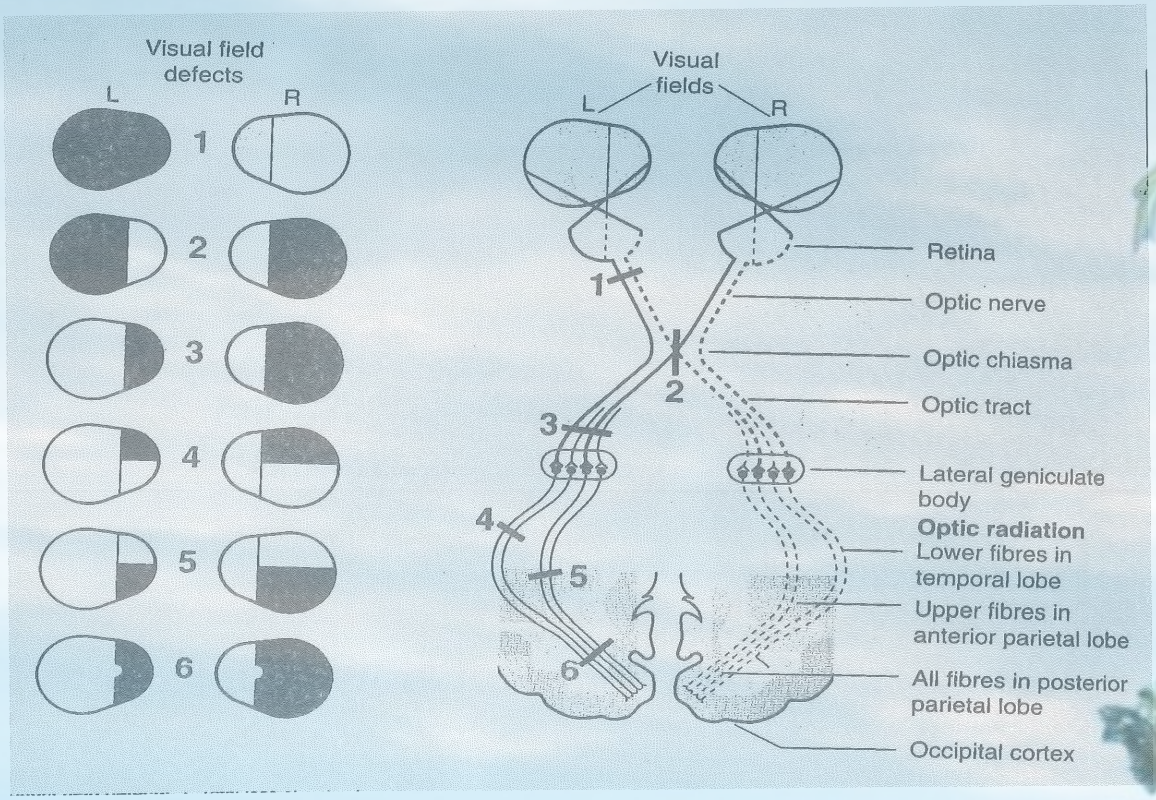
3. **Lesion in the Optic Tract:**
   - Contralateral Homonymous Hemianopia (bilateral half blindness).

4. **Lesion in the Optic Radiation (lower fibres):**
   - Upper quadrantic Contralateral Homonymous Hemianopia.
   - Preservation of the light reflex.

5. **Lesion in the Optic Radiation (upper fibres):**
   - Lower quadrantic Contralateral Homonymous Hemianopia.
   - Preservation of the light reflex.

> **In complete lesion of the Optic Radiation**, there will be:
> - Contralateral Homonymous Hemianopia (bilateral half blindness).
> - Preservation of the light reflex.

6. **Lesion in the Occipital Lobe:**
   - Contralateral Homonymous Hemianopia (bilateral half blindness).
   - Preservation of the light reflex.
   - Preservation of the macular vision (due to double blood supply).



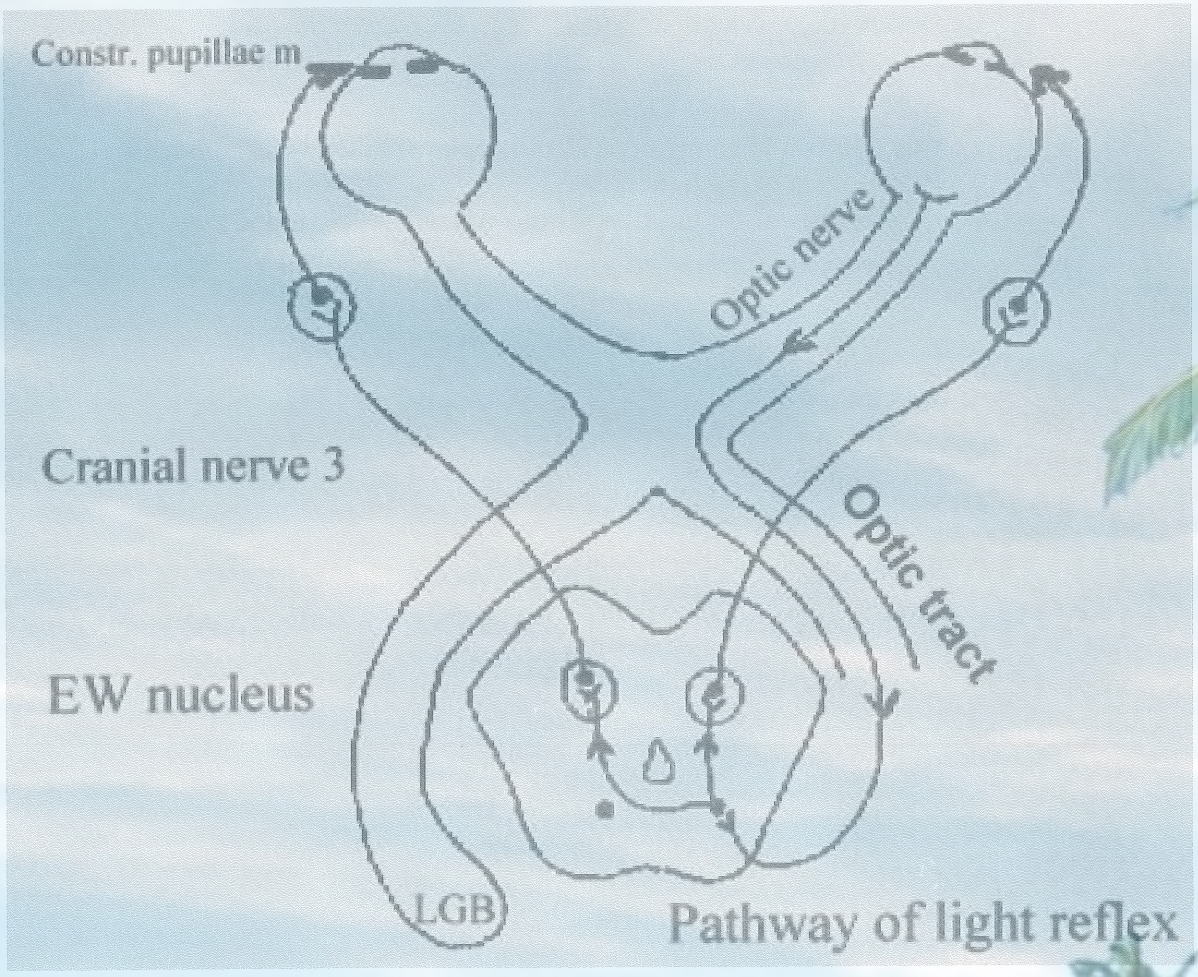
LIGHT REFLEX

Definition

- Exposure of one eye to bright light results in :
  1. Constriction of the same side (direct reflex).
  2. Constriction of the opposite side (consensual reflex).

Pathway

- Impulses pass in the optic nerve, chiasma & then optic tract.
- Fibres do not reach the lateral geniculate body, but pass to the Edinger-Westphal nuclei (EW nuclei) of both sides in the midbrain.
- Fibres then pass from the EW nuclei of both sides to the oculomotor nerves.
- Fibres finally terminate in the constrictor pupillae muscles of both eyes.



3. OCULOMOTOR NERVE

- **Function**
  - Extraocular movements.
  - Elevation of the upper eyelid.
  - Pupillary constriction.

- **Lesion**

A) **External Ophthalmoplegia:**

1. Ptosis.
2. Divergent paralytic squint (the eye looks out & down due to the unopposed action of the lateral rectus " Cr N 6 " & the superior oblique " Cr N 4 ").
3. Diplopia.

B) **Internal Ophthalmoplegia:**

1. Ipsilateral mydriasis.
2. Ipsilateral loss of light reflex: *loss of direct light reflex on the affected eye.*

4. TROCHLEAR NERVE

- **Function**
  - Extraocular eye movement: *inwards & downwards* (superior oblique muscle).

- **Lesion**
  1. Limitation of eye movement on looking *inwards & downwards*.
  2. Diplopia.

6. ABDUCENT NERVE

- **Function**
  - Extraocular eye movement: *outwards* (lateral rectus muscle).

- **Lesion**
  1. Limitation of eye movement on looking *outwards*.
  2. Diplopia.

PUPILLARY DISORDERS

A. Myosis:

1. **Horner's syndrome** (due to sympathetic lesion):
   - Ptosis.
   - Myosis.
   - Enophthalmos.
   - Anhydrosis.

2. **Argyll-Robertson pupil** (due to neurosyphilis, encephalitis, DS, DM):
   - Myotic, irregular, eccentric pupil.
   - Lost light reflex.

   It is due to a lesion in the MB destroying:
   - The sympathetic pupillodilator fibres.
   - The fibres of the light reflex.

3. **Pontine hemorrhage.**
4. **Morphine poisoning.**

} **Pin-point pupil**

B. Mydriasis:

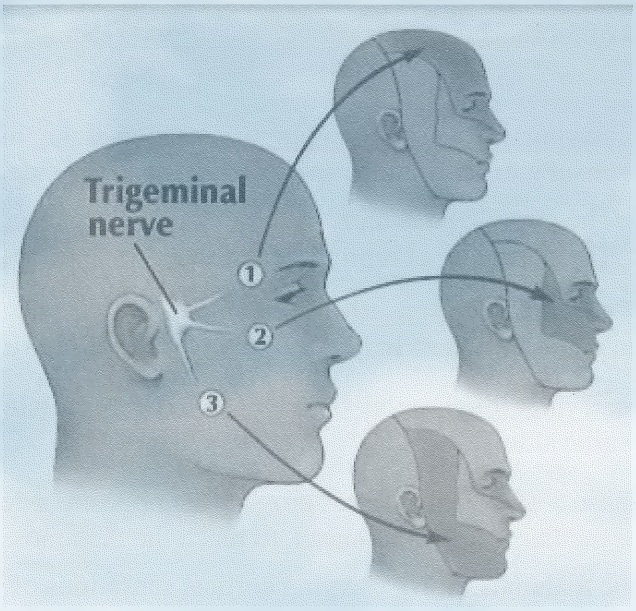
1. **Oculomotor nerve lesion:**
   - Dilated fixed pupil.

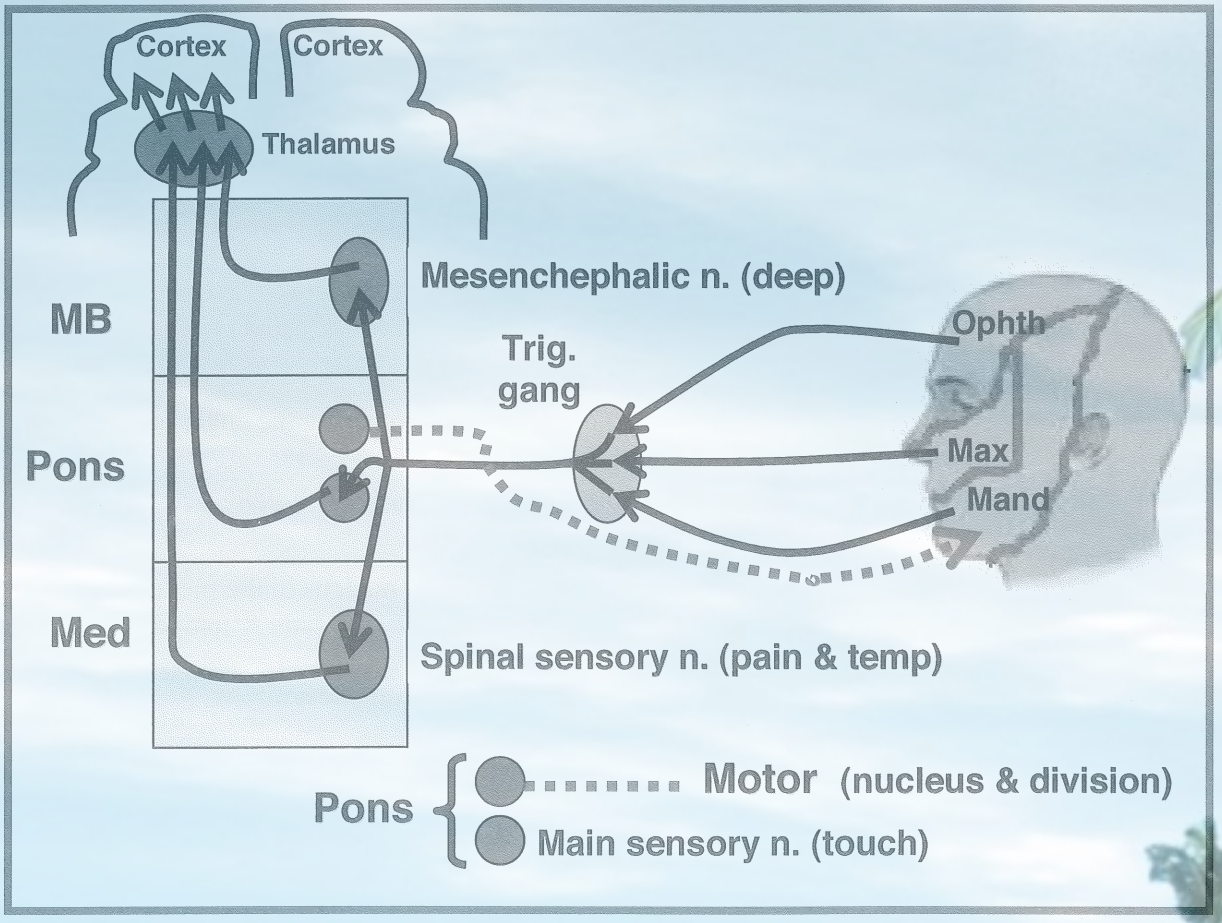
2. **Adie's pupil** (Heredo-familial):
   - Dilated pupil.
   - Lost light reflex.
   - Lost ankle reflex.

   It is Heredo-familial:
   - In females.
   - Unilateral.

5. TRIGEMINAL NERVE

PATHWAY OF THE TRIGEMINAL NERVE





▪ Function

1- The Sensory Division:

- It conducts sensations from the face (except the angle of the mandible, supplied by C_2), the anterior 2/3 s of the tongue & the buccal cavity.
- It is formed of 3 branches: *ophthalmic*, *maxillary*, and *mandibular*.
- Pathway:

  a) Fibres carrying **pain & temperature** sensations relay in the **spinal sensory nucleus** in the **medulla** on the **same side**, where:

  - The upper face is represented in the lower part of the nucleus & vice versa.
  - The outer face is represented in the inner part of the nucleus & vice versa.

  b) Fibres carrying **deep sensations** relay in the **mesencephalic nucleus** in the **midbrain** on the **same side**.

  c) Fibres carrying **touch** relay in the **main sensory neucleus** in the **pons** on the **same side**.

  - From these 3 nuclei, new fibres cross to the opposite side, then ascend with the medial & lateral lemnisci, to reach the thalamus.
  - From the thalamus, fibres carrying cortical sensations of the face ascend to end in the cortical sensory area in the parietal lobe.

2- The Motor Division:

- It arises from the motor nucleus in the pons.
- It supplies the muscles of mastication.

▪ Lesion

1. Sensory affection:

a) Peripheral lesion:
- Loss of sensations on the same side of the face (sparing the angle of the angle of the mandible).
- Loss of general sensations over the anterior 2/3 s of the tongue, (in lesions of the mandibular division).

b) Central lesion: (mainly affects the spinal sensory nucleus in the medulla):
- Ipsilateral dissociated sensory loss of the face, i.e. lost pain & temperature with preservation of touch & deep sensations.
- The clinical picture varies according to the site of the lesion:

▪ Lesion starting from above (lower pontine tumour):	affects the lower part of face.
▪ Lesion starting from below (upper cervical lesions):	affects the upper part of face.
▪ Lesion starting from periphery (Tabes dorsalis):	affects the central part of face.
▪ Lesion starting from midline (syringobulbia):	affects the outer part of face.

2. **Motor affection:**
   a) Weakness of the muscles of mastication on the same side of the lesion.
   b) Deviation of the jaw to the affected side due to the unopposed action of the pterygoid muscles of the healthy side.

3. **Reflex affection:**
   a) Ipsilateral loss of the corneal reflex (afferent 5, efferent 7).
   b) Ipsilateral loss of the palatal reflex (afferent 5, efferent 10).
   c) Appearance of jaw reflex (afferent 5, efferent 5):
   in cases of bilateral UMNL above the pons.

DD OF FACIAL PAIN

1. Trigeminal neuralgia.

2. Post-herpitic neuralgia:

- The pain usually affects the ***ophthalmic division***.
- The pain is followed by herpetic skin eruption.

3. Sinusitis:

- The pain is worse in the morning, with tenderness over the involved sinus.

4. Ocular (glaucoma):

- The pain is behind the eye, with associated visual symptoms.

5. Dental:

- The pain is around the mouth.

6. Cluster headache:

- Refer to the chapter of " Migraine ".

7. Costen's syndrome: (Temporo-mandibular joint dysfunction).

TRIGEMINAL NEURALGIA
(Tic douloureux)

DEFINITION

- Severe attacks of pain along one or more of the sensory branches of the trigeminal nerve, usually the ***maxillary*** or ***mandibular***.

ETIOLOGY

- The exact cause is **UNKNOWN**, but there are some related diseases:
  - Nerve root compression (in the cerebello-pontine angle by):
    - Tumour.
    - Aneurysm.
    - ***Vascular anomaly: Abnormal vascular course of the SCA.*** *
  - DS & DM.

CLINICAL PICTURE

- The "Pain syndrome" is diagnosed by the "History" alone:

  - **Site & reference:**
    - One or more branches of the TN (usually the ***maxillary*** or ***mandibular***).
    - Pain is unilateral (rarely bilateral).

  - **Character & duration:**
    - Severe agonizing: stabbing, burning, electric shocks.
    - Several seconds to several minutes (rarely hours or even longer).
    - Recurrent: but the patient is completely free in between the attacks.

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* *SCA:* Superior cerebellar artery

- **Precipitated by:**
  - Movements of the jaw: e.g. laughing & mastication.

- **Relieved by:**
  - Spontaneously, or by potent analgesics.

- **Associated symptoms:**
  - Pain provokes brief muscle spasm of the facial muscles, thus producing a tic.

TREATMENT

- ***Medical:***
  - Carbamazepine: 500 – 1000 mg / day orally.
  - Analgesics.

- ***Surgical:*** **e.g.**
  - Section of the affected sensory root.
  - **MICROVASCULAR DECOMPRESSION.**

7. FACIAL NERVE

▪ Function

The facial nerve is a mixed nerve, containing: ***motor***, ***sensory*** and ***autonomic*** fibres.

The ***motor*** part: supplies the muscles of expression of the face as well as 4 other muscles,

- Platysma.
- Posterior belly of the digastric muscle.
- Stapedius.
- Stylohyoid.

The ***sensory*** part: receives taste sensations from the anterior 2/3 s of the tongue.

The ***autonomic*** part: supplies the lacrimal, the submandibular & sublingual salivary glands.

▪ Anatomy

1. Anatomy of the motor part: " See figure "

The **motor nucleus** of the facial nerve lies in the **pons**, near the 6th nerve nucleus.
It is controlled by 2 types of supranuclear fibres:

a) Pyramidal Fibres:
   - The upper part of the nucleus is supplied from both pyramidal tracts.
   - The lower part of the nucleus is supplied from the opposite pyramidal tract only.

b) Extra-pyramidal Fibres:
   - For emotional & associated movements.

From the nucleus, the motor fibres form a loop around the 6th nerve nucleus, then pass laterally to emerge at the lower part of the pons.

The nerve runs laterally between the 6th and 8th cranial nerves, in the subarachnoid space of the **cerebellopontine angle** to enter, through the **internal auditory meatus (IAM),** into the facial canal.

In the **facial canal**, the motor part of the facial nerve becomes adherent to its sensory and autonomic parts. It gives the **nerve to Stapedius muscle**.

Finally, the facial nerve leaves the canal through the **stylomastoid foramen SF**, passes through the **parotid gland** to divide into its terminal branches to innervate the muscles of the face.

2. Anatomy of the sensory and autonomic parts: " See figure "

- In the facial canal lies the **geniculate ganglion**.

This ganglion gives 2 branches, **a peripheral branch** & **a central branch**.

a. The peripheral branch: runs laterally and divides into the greater superficial petrosal nerve and the chorda tympani.

- The **greater superficial petrosal GSP** nerve passes forwards to relay in the sphenopalatine ganglion where a new set of fibres gives autonomic supply to the lacrimal gland.

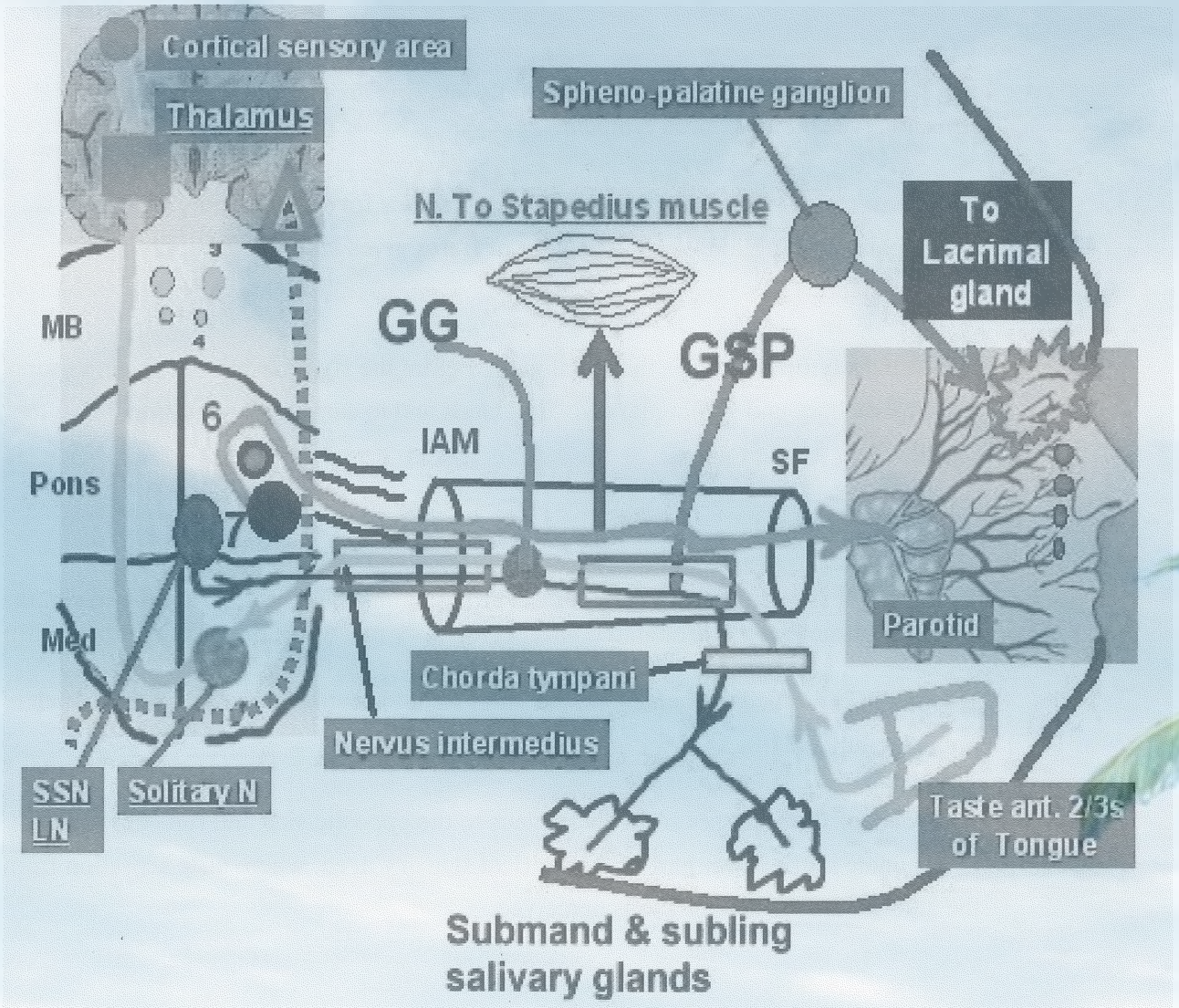
- The **chorda tympani** leaves the facial nerve before the stylomastoid foramen, to supply the submaxillary and sublingual salivary glands and to carry taste sensations from the anterior 2/3s of the tongue.

b. The central branch: passes centrally, joins the motor part of the nerve, then enters the cranial cavity through the internal auditory meatus as the **nervus intermedius.**

- This nervus intermedius then enters the brain stem to terminate in the **solitary nucleus** in the medulla.

- A new set of fibres passes from the nucleus to the opposite side and runs upwards to terminate in the lower part of the **cortical sensory area**, where taste sensation from the anterior 2/3s of the tongue is perceived.

PATHWAY OF THE FACIAL NERVE



▪ Lesions of The Facial Nerve

The lesion may be:

1. UMNL: (Supra-nuclear lesions)

- Lesions of the cortico-bulbar tract (pyramidal tract) at any point from:

  The motor area in the cortex → to the facial nucleus in the pons.

2. LMNL: (Nuclear & infra-nuclear lesions)

- Lesions that affect the facial motor nucleus or the nerve itself.

1. UMNL: (SUPRA-NUCLEAR LESIONS)

- ***Causes:***

1.	Vascular:	*Embolism, Thrombosis, Hemorrhage.*
2.	Traumatic:	Head injury.
3.	Inflammatory:	Post-encephalitic.
4.	Neoplastic:	Glioma.
5.	Demyelinating:	DS.
6.	Degenerative:	MND.

- ***Clinical features:***

Features of UMN Facial paralysis:

Paralysis of the LOWER HALF of the face on the OPPOSITE SIDE of the lesion:

- Dropping of the angle of the mouth.
- Deviation of the mouth to the healthy side on showing the teeth.
- Dribbling of saliva.
- Flattening of the naso-labial fold.
- Inability to blow the cheek.
- Accumulation of food behind the cheek.

2. LMNL: (NUCLEAR & INFRA-NUCLEAR LESIONS)

- ***Causes:***

1. Pontine Lesions. } Nuclear Lesions

2. Cerebello-Pontine Angle
3. Facial Canal Lesions. } Infra-nuclear Lesions
4. Extracranial Lesions.

- ***Clinical features:***

1. Features according to the level of the lesion (see later: *).

2. **Features of LMN facial paralysis**:

Paralysis of: ALL MUSCLES of the face on the SAME SIDE of the lesion:

- Dropping of the angle of the mouth.
- Deviation of the mouth to the healthy side on showing the teeth.
- Dribbling of saliva.
- Flattening of the naso-labial fold.
- Inability to blow the cheek.
- Accumulation of food behind the cheek.

PLUS

- Inability to raise the eye brows.
- Inability to close the eyes.
- Absence of wrinkles of the forehead on looking upwards.

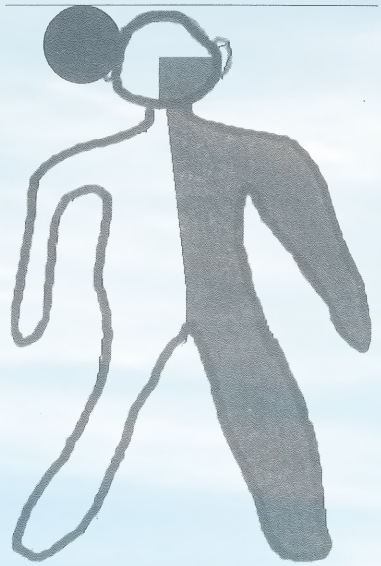
* LOCALISATION OF THE SITE OF LMNL

SITE OF LESION	CAUSES
Pontine Lesions (Nuclear Lesions) 1. Paralysis of facial muscles. 2. No impairment of taste sensation. 3. No impairment of salivation nor lacrimation. 4. May be other LMN cranial palsies on the same side or hemiplegia on the opposite side.	1. Vascular: - Vetebro-basilar insufficiency. - Millard-Gubler syndrome. 2. Infective: - Encephalitis, Poliomyelitis. 3. Neoplastic: - Glioma. 4. Demyelinating: - DS.
Cerebello-Pontine Angle Lesion (ALL) 1. Paralysis of facial muscles. 2. ↓ taste on anterior 2/3s of tongue. 3. ↓ salivary & lacrimal secretions. 4. Associated Cr. 5, 6 & 8 palsies on same side. 5. Ipsilateral cerebellar ataxia.	1. Infective: - Basal meningitis. 2. Neoplastic: - Acoustic neuroma, Meningioma.
Facial Canal Lesion (If) 1. Paralysis of facial muscles. 2. ↓ taste on anterior 2/3s of tongue, and ↓ salivation if chorda tympani is involved. 3. Diminished lacrimation if the greater superficial petrosal nerve is involved. 4. Hyperacusis if the nerve to Stapedius is involved.	1. Traumatic: - Fracture base. 2. Infective: - Otitis media, Herpes zoster. 3. Neoplastic: - Facial neuroma. 4. Bell's palsy.
Extracranial Lesions (After its exit from the stylomastoid foramen) Paralysis of facial muscles ***only***.	1. Neuropathy: - Diabetic, Leprotic. 2. Myopathy: - Facioscapulohumeral. 3. Myasthenia. 4. Invasion by a tumour: e.g. *from parotid*. 5. Injury to the nerve during surgery.

Differentiation Between UMNL & LMNL of the Facial Paralysis

UMNL	LMNL
1. Affects the Pyramidal tract above the Facial Nucleus.	1. Affects the Facial Motor Nucleus, or, the Nerve itself.
2. Paralysis of the muscles of LOWER HALF of the face on the OPPOSITE SIDE of the lesion (supplied from the opposite pyramidal only).	2. Paralysis of ALL MUSCLES of the face (upper & lower halves), on the SAME SIDE of the lesion).
3. Paralysis involves the voluntary movement, BUT: spares the emotional & associated movements (supplied from the extra-pyramidal fibres).	3. Paralysis involves the voluntary, emotional and associated movements.
4. Paralysis is associated with Hypertonia and Hyperreflexia.	4. Paralysis is associated with Hypotonia and Hyporeflexia.
5. There is associated Hemiplegia on the SAME SIDE of the facial paralysis.	6. If there is Hemiplegia, it is on the OPPOSITE SIDE of the facial paralysis (crossed Hemiplegia).

Lesion (Right)



Lesion (Right)

Bell's Palsy

DEFINITION

- It is an acute LMN paralysis of the face, due to a non-suppurative inflammation and oedema of the facial nerve in the FACIAL CANAL near the stylomastoid foramen.

- It is usually unilateral, may be recurrent & sometimes runs in families.

ETIOLOGY

- UNKNOWN: *Many causes have been suggested:*

1. **Air drafts:** Exposure to air drafts may lead to *inflammation*, *oedema*, & *compression* of the nerve at the stylomastoid foramen.
2. **Viral infection:** It may be secondary to a neurotropic virus (*Herpes zoster*), *Herpes simplex*.
3. **Autoimmune:** There is evidence of high levels of *immunoglobulins* in the patient's serum.

CLINICAL PICTURE

1. PAIN:
   - The entire course of the disease may be PAINLESS, *OR*,
   - The disease may also present with acute PAIN behind the ipsilateral ear.
2. PARALYSIS: *" one or two days after the pain "*
   - Complete paralysis of the facial muscles on the affected side of LMN features, (features of FACIAL CANAL lesion).

COMPLICATIONS

1. CROCODILE TEARS:
   - *Excessive tears:* flow from the affected eye during eating.
   - *Explanation:* the regenerating fibres to the submandibular salivary glands become misdirected and reach the lacrymal gland.
2. CORNEAL ULCERS.
3. INCOMPLETE RECOVERY:
   - This may result in residual facial asymmetry.

TREATMENT

1. REASSURANCE.

2. PHYSIOTHERAPY:

a. Massage of the facial muscles.
b. Infrared irradiation to the face.
c. Galvanic stimulation to the facial muscles.

3. MEDICAL TREATMENT:

a. *Corticosteroids:* Oral prednisolone (1 mg / Kg / day): to ↓ inflammation & oedema.

b. *Corneal protection:* Eye ointment during sleep, sunglasses during daytime.

4. SURGICAL TREATMENT:

a. For Facial nerve palsy: Decompression of the facial nerve ***or*** Facial nerve grafting.
b. For Crocodile tears: Section of the regenerating fibres.
c. For Residual asymmetry: Plastic surgery.

PROGNOSIS

- Most patients (80%) recover in few weeks.
- The remaining (20%) need surgical interference.

8. COCHLEO-VESTIBULAR NERVE

It has 2 divisions:

1. **Cochlear division:**
   - **Function:**
     - Hearing.
   - **Lesion:**
     - Irritative: Tinnitus.
     - Destructive: Deafness.
2. **Vestibular division:**
   - **Function:**
     - Equilibrium.
   - **Lesion:**
     - Vertigo.
     - Nystagmus.
     - Incoordination in the same side of the body.

Vertigo

DEFINITION

- **It is the sense of rotation of the body in steady surroundings or the reverse i.e.:**
  - The body may be felt to rotate or fall while the surroundings are steady, or
  - The surroundings themselves appear to rotate around the body.
- **It is aggravated by movements of the head and it persists in all positions:** *Sitting, Standing* or *Supine.*
- **It is usually associated with autonomic manifestations in the form of:** *Nausea, vomiting, pallor, sweating.*
- It should be differentiated from dizziness, where the unsteadiness is not associated with a sense of rotation.

ETIOLOGY

1) **Labyrinthine:**
   a) Physiological: sea sickness, car sickness.
   b) Pathological: labyrinthitis, Meniere's disease.

2) **Peripheral nerve:**
   - Cerebello-pontine angle lesion as acoustic neuroma.
   - Vestibular neuritis.

3) **Brain stem:**
   - Vertebro-basilar artery insufficiency.
   - Posterior inferior cerebellar artery occlusion: PICA.
   - Disseminated sclerosis: DS.
   - Encephalitis.

4) **Cerebral:**
   - Increased intracranial tension.

5) **Psychogenic:**
   - It occurs in acute anxiety . It responds to tranquillisers.

6) **Benign Positional Vertigo:**
   - It is usually idiopathic *or* due to inner ear disease.
   - It is associated with nystagmus & is usually by changing the position such as turning in bed.
   - It is usually mild, transient, lasting less than 30 seconds.

TREATMENT

1) Treatment of the cause.
2) Cinnarizine (Stugeron) and Dramamine may be used.

Bulbar Palsy

DEFINITION

- Paralysis of the muscles supplied by the cranial nerves arising from the Medulla: 9 , 10 , 12.

CLINICAL PICTURE

1. Dysarthria.
2. Dysphagia.
3. Hoarseness of voice.
4. Nasal regurgitation of food.

TYPES

I. True Bulbar Palsy:

- Bilateral LMNL of the cranial nerves in the Medulla.
- Common causes are:
  1. Poliomyelitis.
  2. MND.
  3. Syringobulbia.

II. Pseudo Bulbar Palsy:

- Bilateral UMNL of the cranial nerves in the Medulla.
- Common causes are:
  1. DS.
  2. MND.
  3. Brain stem tumours.

	True Bulbar Palsy	Pseudo Bulbar Palsy
1. Palatal reflex	Absent	Exaggerated
2. Pharyngeal reflex	Absent	Exaggerated
3. Jaw reflex	Absent	Present
4. Quadriplegia	Absent	Present
5. Tongue	- Wasting - Flacidity - Fasciculations	- No wasting - Spasticity - No fasciculations

CEREBRO-VASCULAR DISEASE

- Diseases of the BRAIN VESSELS include:

1. **STROKE.**
2. **TRANSIENT ISCHEMIC ATTACKS (TIAs).**

STROKE

DEFINITION

" Brain Attack "

- ACUTE INJURY to the brain due to a VASCULAR problem.
- It produces neurological deficits:
  - *Acute in onset.*
  - *Persistent for more than 24 hours.*

ETIOLOGY

1. **Ischemic stroke:** " The most common cause " **85 %**

   OCCLUSION of a blood vessel to a part of the brain → Ischemia.

   - Thrombosis on top of cerebral atherosclerosis: *Acute occlusion.*
   - Embolism: *Sudden occlusion.*

2. **Hemorrhagic stroke:** " Less common cause " **15 %**

   RUPTURE of a blood vessel in a part of the brain → Bleeding.

1. Thrombosis: *"clot in situ resulting in cerebral infarction"*

1. ***Vessel wall diseases:*** *e.g.*
   - Cerebral atherosclerosis: **THE MOST IMPORTANT**
   - Vasculitis: *PAN & SLE.*
2. ***Blood diseases causing hyperviscosity:*** *e.g.*
   - Polycythemia (↑ RBCs).
   - Leukemia (↑ WBCs).
   - Thrombocytosis (↑ Platelets).
3. ***Circulation diseases:*** **SLOW CIRCULATION** *e.g.*
   - Heart failure.
   - Systemic hypoperfusion, e.g. *Dehydration & shock.*

2. Embolism: *"wandering clot resulting in cerebral infarction"*

The source of the embolus may be:

1. ***Heart:*** *" The most common source "*
   - MS with AF, Vegetations of SBE, Mural thrombus in AMI.
2. ***Distal vessels:***
   - Arterial: Detached atheromatous plaque from carotid vessels.
   - Venous: DVT → detached thrombus → paradoxical embolism through VSD or ASD.

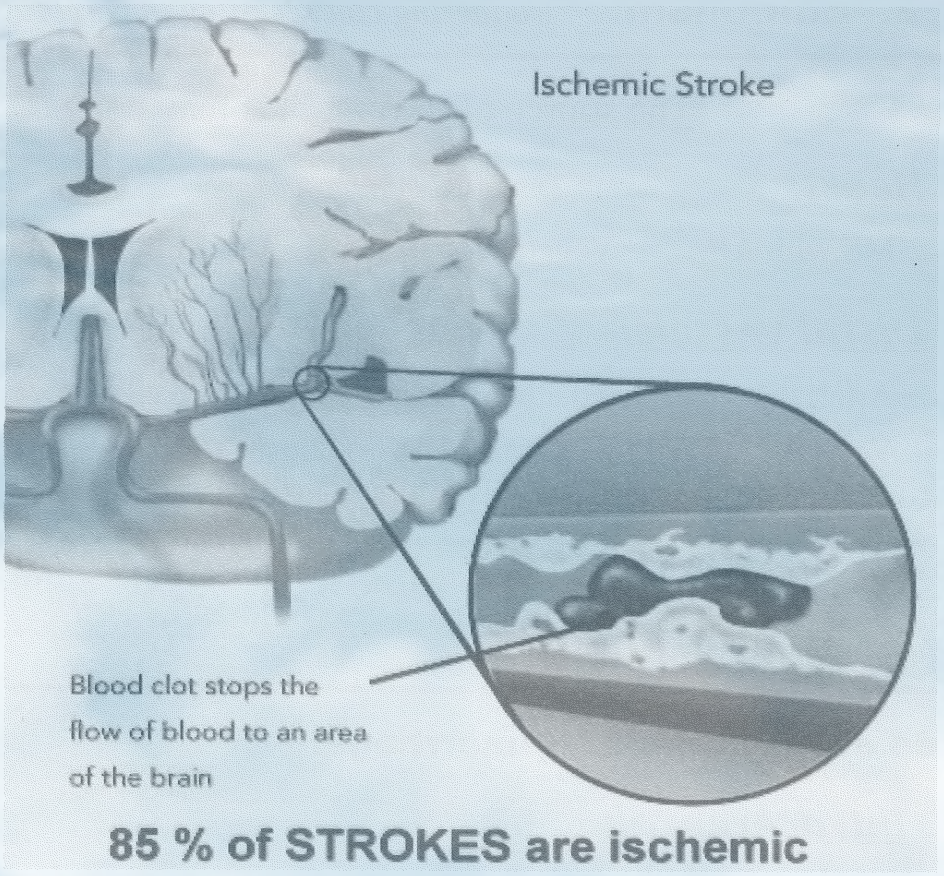
3. Hemorrhage:

Types of Intracranial Hemorrhage:

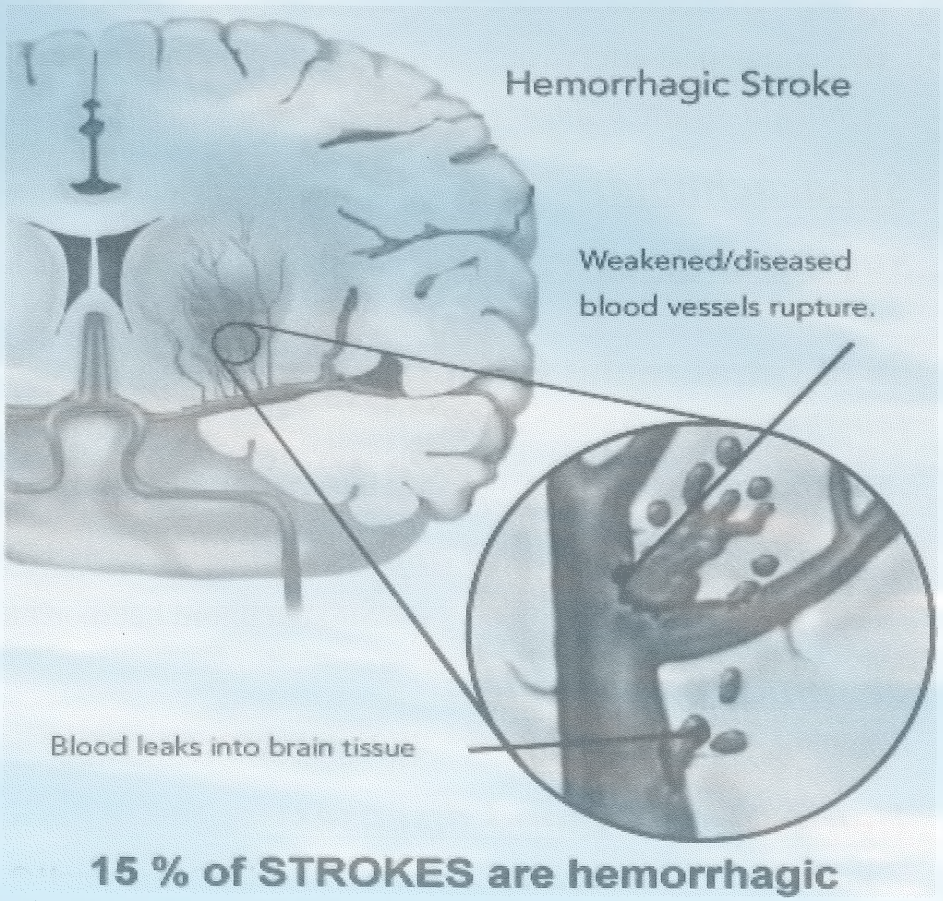
1. ***Intracerebral:***
   - The bleeding is in the brain substance & may leak into the ventricles.
   - This is very serious as the blood may compress vital centres.
   - The commonest artery causing intracerebral hemorrhage is the *"lenticulo-striate branch* of the *middle cerebral artery"*
2. ***Subarachnoid:*** Bleeding is in the subarachnoid space.
3. ***Subdural or extradural:*** Bleeding often forms a hematoma.

Causes of Intracranial Hemorrhage:

1. ***Hypertension:***
   The commonest cause of: Intracerebral haemorrhage.
2. ***Rupture of intracranial aneurysm, or A-V malformation:***
   The commonest cause of: Subarachnoid haemorrhage.
3. ***Trauma to the head:***
   The commonest cause of: Subdural haematoma.
4. ***Hemorrhagic blood diseases:***
   Purpura & Hemophilia.
5. ***Anticoagulants.***



85 % of STROKES are ischemic



15 % of STROKES are hemorrhagic

RISK FACTORS

1. Non – modifiable:

- Age: old.
- Sex: male : female = 3 : 1.
- Type A personality : nervous, intellectual.
- Genetic factors: positive family history.

2. Modifiable:

a) *High risk*:

- **H**eart diseases: *especially valvular heart diseases & AF.*
- **H**yperlipidemia: ↑ total cholesterol, ↑ LDL, ↓ HDL.
- **HYPERTENSION**: causes endothelial damage.
- Diabetes mellitus.
- Cigarette smoking.

b) *Less risk*:

- Obesity.
- Diet: rich in saturated fat & cholesterol.
- Physical inactivity.
- Psychological stress.
- *H*yperuricemia.
- *H*omocysteinemia.
- *H*eavy alcohol intake.
- CCPs.

CLINICAL PICTURE

- Neurological deficits commonly produced depend on the affected brain area:

1. **HEMIPLEGIA.**
2. **HEMIANESTHESIA.**
3. **Speech *problems*:** Aphasia or Dysarthria.
4. **Vision *problems*:** especially field defects.
5. **Ataxia:** Lack of co-ordination or Lack of balance.
6. **Cranial nerve paralysis.**

CLINICAL TYPES

1. **Stroke in evolution:** **(evolving stroke)**

   An enlarging brain infarct with neurologic deficits that worsen over 24 to 48 h.

2. **Completed stroke:**

   A stable brain infarct with neurologic deficits that signify STABLE injury.

INVESTIGATIONS

I. BRAIN IMAGING

a) CT scan

1. Ischemic stroke:
   - Infarctions may not appear until 24 hours after presentation.
   - Small infarctions may not appear at all.

2. Hemmorrhagic stroke:
   - Bleeding appears immediately (even very small lesions).
   - *Therefore:* CT scan can help to rule out a hemorrhagic stroke.

b) MRI

- It provides a more detailed and sensitive picture of the brain.

II. OTHER IMAGING

a) CARDIAC IMAGING: CXR, ECG, Echocardiography.

b) VASCULAR IMAGINMG: DOPPLER ultrasonic imaging.

III. LABORATORY TESTS

a) CBC.

b) PT & APTT.

c) ***Risk factors***: e.g. blood glucose, lipid profile, UA, homocysteine.

d) ***Hypercoagulability:*** e.g. protein C, protein S, Antithrombin III.

TREATMENT

I. GENERAL CARE: " For: Hemiplegic patient or a Comatosed patient "

" Stroke Unit "

1. **Skin:**
   - Frequent change of the patient's position (every 2 hours), and of the bed sheets.
   - Frequent wash of the back & pressure points by alcohol followed by powder.
2. **Swallowing & nutrition:**
   - Tube feeding. & IV fluids.
3. **Balance of fluids:**
   - Controlled IV fluids.
4. **Breathing:**
   - Suction of secretions.
   - Oxygen inhalation.
5. **Bladder:**
   - Catheterisation & urinary antiseptics.
6. **Bowels:**
   - Daily enema.

II. SYMPTOMATIC:

1. **EARLY:**
   - Cerebral dehydrating agents (Mannitol or Corticosteroids): to ↓ brain oedema.
   - Prophylaxis against STRESS ULCER: "Refer to Cardiology".
2. **LATE:** **PHYSIOTHERAPY.**
3. **ALL through:** **Vitamin and tonics.**

III. SPECIFIC: " TTT of the cause "

A) Thrombosis:

1. **Control of Blood Pressure.**

2. **Thrombolytic therapy:** using **IV tPA** may be of benefit.

3. **Antiplatelets:** ***because platelet aggregation is increased after thrombosis:***

- ***Aspirin*** *(low dose):* *75 – 300 mg / day (single dose).*
- *Dipyridamole:* *75 mg twice daily.*
- *Ticlopidine:* *250 mg twice daily.*
- ***Clopidogrel:*** *75 mg once daily.*

4. **Anticoagulants:** ***They are not used in all cases:***

Indications

1. Stroke in evolution: ***i.e.*** gradual progressive weakness (over days) denoting gradual & progressive thrombus formation.
2. Recurrent TIAs.
3. Embolic hemiplegia: esp. in cardiac cases to prevent recurrent embolisation.

Contraindications

1. Haemorrhagic infarction; it is excluded by using the CT scan.
2. Haemorrhagic blood diseases.
2. Hepatic & Renal failure.
3. Subacute bacterial endocarditis (SBE).

5. **Other drugs:**
   - Trivastal or Trental: may ↑ the cerebral blood flow.

B) Embolism:

1. **Treatment of the source of emboli:** *e.g. MS with AF.*

2. **Anticoagulants:** *esp. in cardiac cases to prevent recurrent embolisation.*

C) Hemorrhage:

1. **Control of Blood Pressure:**
   - Refer to: Hypertensive emergencies in Cardiology.

2. **Antifibrinolytic drugs:** e.g. amino caproic acid
   - They delay clot lysis & thus prevent rebleeding, However: They may ↑ cerebral ischemia.

3. **Surgery:**
   - Evacuation of the haematoma provided it is not intraventricular.

TRANSIENT ISCHEMIC ATTACKS (TIAs)

DEFINITION "Mini – Stroke"

"Sudden attacks of temporary cerebral ischemia too short to cause infarction"

- DURATION: few minutes or hours (maximum 24 hours) followed by ***complete recovery***.
- VALUE: A TIA is a ***warning*** for an approaching ischemic stroke.

- If the attack lasts over 24 hours & then the patient recovers, it is called: **RIND:**
  " Reversible Ischemic Neurological Deficit "

ETIOLOGY

- Temporary reduction or cessation of cerebral blood flow in a specific neurovascular distribution can be due to *low flow through a partially occluded vessel* or to *an acute thromboembolic event*.

1. Most commonly: *cerebral emboli arising from:*
   - Ulcerated atherosclerotic plaques: in the carotid or vertebral arteries.
   - Thrombi: in a diseased heart, e.g. AF.
2. Less commonly:
   - *Vasculitis* (e.g. SLE & PAN), & *Hyperviscosity* (e.g. Polycythemia).

CLINICAL PICTURE

1. **CAROTID TIAs.** **(see later)**
2. **VERTEBRO-BASILAR TIAs.** **(see later)**

PROGNOSIS

TIA is a warning for an approaching ischemic stroke. If the time period of blood supply impairment lasts more than a few minutes, the nerve cells of that area of the brain die and cause permanent neurologic deficit.

One third of people who had a TIA will develop a stroke.

Risk of developing stroke is stratified according to the **A B C D²** score

A **A**ge ≥ 60 years = 1 point.

B **B**P ≥ 140 / 90 mmHg = 1 point.

C **C**linical features:

- Unilateral weakness = 2 points.
- Speech disturbance without weakness = 1 point.

D **D**uration of the attack:

- ≥ 60 minutes = 2 points.
- 10 to 59 minutes = 1 point.

D **D**iabetes = 1 point.

Interpretation of score: Score / Risk ratio

- Score 1 – 3: (low).
- Score 4 – 5: (moderate).
- Score > 6: (high).

- **Important question:** **"Self – assessment"**

"What is the DD of TIA" ??

INVESTIGATIONS

I. BRAIN IMAGING

1. Cerebral angiography.
2. CT scan: to rule out intracranial hemorrhage.

II. OTHER IMAGING

1. CARDIAC IMAGING: CXR, ECG, Echo (TTE, TEE).
2. VASCULAR IMAGINMG:
   - DOPPLER ultrasonic imaging.
   - MRA.

III. LABORATORY TESTS

1. CBC.
2. PT & APTT.
3. ***Risk factors***: e.g. blood glucose, lipid profile, uric acid, homocysteine.
4. ***Hypercoagulability:*** e.g. protein C, protein S, Antithrombin III

TREATMENT

A) Medical:

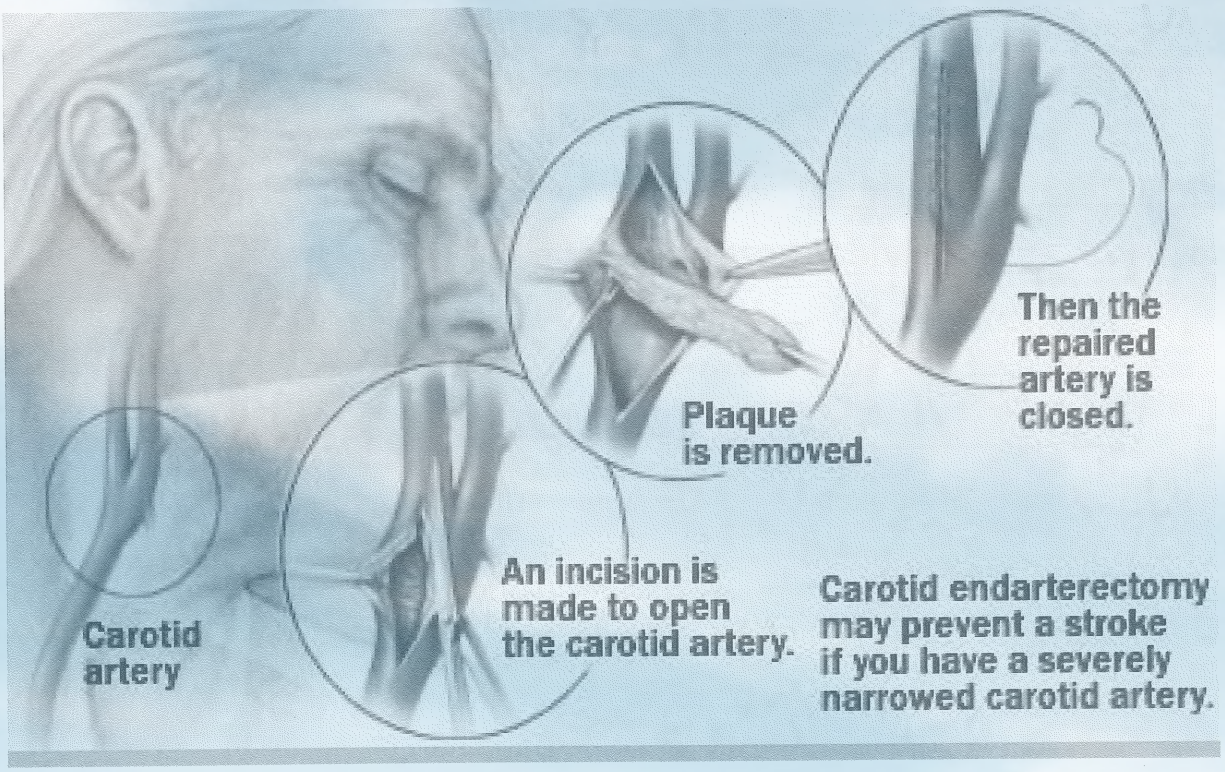
1. Antiplatelets:
   - ***Aspirin*** *(low dose):* *75 – 300 mg / day (single dose).*
   - *Dipyridamole:* *75 mg twice daily.*
   - *Ticlopidine:* *250 mg twice daily.*
   - ***Clopidogrel:*** *75 mg once daily.*
2. Anticoagulants: e.g. Dindivan.
3. Other drugs: e.g. **Tr**ental or **Tr**ivastal.
4. Treatment of risk factors. e.g. proper control of HTN.

B) Surgical:

1. ENDARTRECTOMY: in carotid artery stenosis of more than 70 %.

Technique: An incision is made to open the artery, the plaques are removed, and the artery is closed.

Value: This preventive surgery clears carotid arteries of fatty deposits (atherosclerotic plaques) before another TIA or stroke occurs.



2. Surgical BYPASS:
   - Bypass between the ECA & MCA is generally not beneficial.

C) Carotid angioplasty & Stent:

Technique:

- Angioplasty: Introduction of a balloon to dilate the stenotic artery.
- Stent: Introduction of an intra-luminal Stent to maintain patency of the dilated artery.

Value:

- It maintains patency in the carotid circulation.
- It may offer benefits over surgical procedures.

HEMIPLEGIA

DEFINITION

Paralysis of one side of the body.

ETIOLOGY

Pyramidal tract lesion at any point from its origin in the cerebral cortex, down to the **5th cervical segment** of the spinal cord.

I. **VASCULAR:** **(STROKE)** ***The most common***

- Causes of Stroke: *Ischemic* (Thrombosis, Embolism), *Hemorrhagic.*

II. **INFECTIVE:** Encephalitis, Meningitis, Brain abscess.

III. **NEOPLASTIC:** Glioma, Meningioma.

IV. **DEMYELINATING:** Disseminated Sclerosis (DS), DEM.

V. **TRAUMATIC:** Cerebral laceration, Subdural haematoma.

VI. **CONGENITAL:** Cerebral palsy.

VII. **HYSTERICAL:** Absence of organic pyramidal lesion.

CLINICAL PICTURE "GENERAL & SPECIFIC"

1. GENERAL CLINICAL PICTURE

Onset & Course

"*Vary according to the lesion*"

- Acute onset & variable course: Vascular, Infective, Traumatic lesions.
- Gradual onset & progressive course: Neoplastic lesions.
- Remittent & Relapsing course: Demyelinating lesions (DS).

Symptoms & Signs

"*Vary according to the onset*"

1) Acute lesions: the hemiplegia passes through 2 stages:
   I. Stage of **flaccidity:** this is due to neuronal shock.
   II. Stage of **spasticity;** this is the stage of established hemiplegia.
2) Gradual lesions: the hemiplegia passes directly to the stage of spasticity.

I. Stage of Flaccid Paralysis: (Shock Stage)

1. On the paralysed side there are:
   - *Muscle tone:* *Completely lost.* (flaccidity)
   - *Deep reflexes:* *Absent.*
   - *Plantar reflex:* *Absent.* (no Babinski sign)

DURATION: 2 – 6 weeks

2. If the onset is associated with coma, the paralysed side is determined by:
   - *Muscle tone asymetry.*
   - *Deep reflex asymmetry.*
   - *Facial asymetry.*

   } **Lateralizing signs**

3. Finally, RECOVERY from the shock stage occurs:
   - *Muscle tone:* *Reappearance, then gradual increase.*
   - *Deep reflexes:* *Reappearance, then gradual increase.*
   - *Plantar reflex:* *Positive* Babinski sign.

This means that the Stage of spasticity starts

II. Stage of Spastic Paralysis: (Stage of established hemiplegia)

1. *Paralysis of one side of the body:* "Paralysis shows a pyramidal distribution"

- Affects the **progravity (weak muscles)** more than the **antigravity (strong muscles)**.

In UL: the extensors (progravity) are weaker than the flexors
In LL: the flexors (progravity) are weaker than the extensors

- Affects the **distal** more than the **proximal** muscles.

The hand is weaker than the shoulder
The foot is weaker than the hip……

2. *Hypertonia (spasticity) of the paralysed side of clasp-knife type:*

- Affects **antigravity (stronger muscle tone)** more than **progravity (weaker muscle tone).**

In UL: the flexors (antigravity) are more spastic than the extensors
In LL: the extensors (antigravity) are more spastic than the flextors

3. *Exaggerated deep reflexes:*

- Deep reflexes in both UL & LL are exaggerated on the paralysed side.
- Pathological deep reflexes (normally absent) may appear.
- Clonus may be present.

4. *Lost superficial reflexes.*

5. *Extensor Plantar reflex.* *(Positive Babinski sign)*

6. *Gait:* *(Circumduction)*

If the patient can walk, his gait is *circumduction* due to:
spasticity of the extensors of LL.

2. SPECIFIC CLINICAL PICTURE

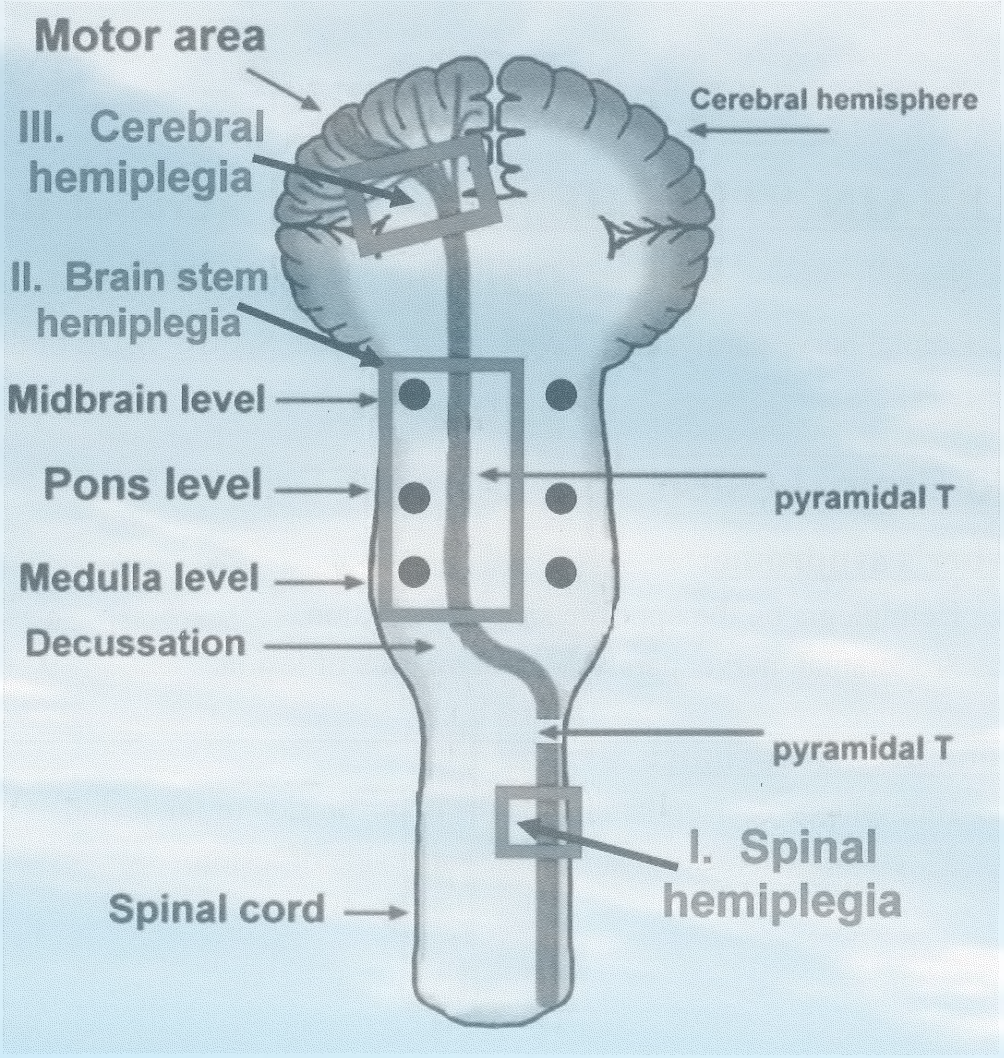
It varies according to:

A. SITE OF LESION B. CAUSE OF LESION

A. ACCORDING TO THE SITE OF THE LESION

The lesion causing hemiplegia may occur at 3 main levels: **" LEVELING "**

I. Spinal cord II. Brain stem III. Cerebral



I. SPINAL HEMIPLEGIA (Brown-Sequard Syndrome)

- The lesion is on one side of the cord & exists between Cl & C5 segments.
- It is caused by: *stab wound, disc prolapse, DS* or *tumour.*

a) At the Level of the Lesion:

1. Ipsilateral ***localised LMNL*** of the muscles supplied by the affected segments.
2. Ipsilateral ***loss of all sensations*** in the area supplied by the posterior roots of the affected segments.

b) Below the Level of the Lesion:

1. Ipsilateral hemiplegia *(UMNL).*
2. Ipsilateral ***deep sensory loss.***
3. Contralateral superficial sensory loss for pain & temperature.
4. Touch diminishes on both sides.

II. BRAIN STEM HEMIPLEGIA (Crossed Hemiplegia)

1. Hemiplegia on the OPPOSITE SIDE of the lesion.
2. Cranial nerve paralysis of LMN nature on the SAME SIDE of the lesion.

1. Mid-Brain Lesion

a) Weber's syndrome:

1. Hemiplegia on the opposite side of the lesion.
2. 3rd cranial nerve paralysis on same side of lesion.

b) Benedict's syndrome:

1. Hemiplegia & Hemiataxia on the opposite side of the lesion.
2. 3rd cranial nerve paralysis on the same side of lesion.

2. Pontine Lesion

a) **Millard Gubler Syndrome:**

1. Hemiplegia on the opposite side of the lesion.
2. 6^{th} & 7^{th} cranial nerve paralysis on the same side of lesion.

b) **Foville Syndrome:**

1. Hemiplegia on the opposite side of the lesion.
2. Loss of conjugate deviation of the eyes to the same side of the lesion due to lesion in the medial longitudinal bundle (**MLB**).

3. Medullary Lesion

a) **Avellis Syndrome:**

1. Hemiplegia on the opposite side of the lesion.
2. 9^{th} & 10^{th} cranial nerve paralysis on the same side of the lesion.

b) **Jackson's Syndrome:**

1. Hemiplegia on the opposite side of the lesion.
2. 10^{th} & 12^{th} cranial nerve paralysis on the same side of the lesion.

III. CEREBRAL HEMIPLEGIA

1. Hemiplegia **plus** UMNL 7^{th} & 12^{th} cranial nerves on the opposite side of the lesion.
2. Absence of any cranial nerve paralysis on the same side of the lesion.

1. Cortical: *"characterised by one or more of the following"*

1. Coma:	if the lesion is extensive.
2. Convulsions:	if the lesion is irritative.
4. Contralateral cortical sensory loss:	if the lesion involves the parietal lobe.
4. Homonymous hemianopia:	if the lesion involves the occipital lobe.
5. Aphasia and agraphia:	if the lesion is in the dominant hemisphere.

6. The paralysis usually involves one limb (monoplegia) especially in vascular lesions.

2. Subcortical:

- *It is similar to cortical hemiplegia except that the paralysis is **more extensive**.*

3. Capsular: *"characterised by the following"*

1. **Complete** hemiplegia associated with UMNL 7th & 12th cranial nerves:
   "All are on the opposite side of the lesion".
2. Hemihyposthesia on the opposite side of the lesion.
3. Hemianopia may occur: (affection of fibres of the optic radiation in the capsule).
4. No **convulsions,** No **aphasia,** No **coma.**

B. ACCORDING TO THE CAUSE OF THE LESION

- The most common cause of hemiplegia is: VASCULAR "STROKE",
  Therefore the DD between: *thrombosis*, *embolism* & *hemorrhage* is important.

Feature	Thrombosis	Embolism	Hemorrhage
1.Age	Old age	Any age	Commonly old age
2.Onset	Rapid (taking hours)	Sudden (taking sec.)	Dramatic or apoplectic
3.Prodromata	TIAs	TIAs	Absent
4.Vomiting	Absent	Absent	Common
5.Consciousness	Usually preserved	Usually preserved	DCL up to deep coma
6.Convulsions	May occur	May occur	Frequent
7.Pupils	Normal and equal	Normal and equal	Dilated and irreactive
8.Fever	Absent	Absent	Present
9.Blood pressure	May be high	Normal	Usually high
10. Heart	May be CAD	Usually valvular lesion	LV hypertrophy
11. CSF	Clear	Clear	Bloody, ↑ tension
12. CT scan, MRI	D I A G N O S T I C		

Hysterical hemiplegia

- Usually: young neurotic ♀.
- The cause: psychological & there is no organic lesion.
- Incidence: usually occurs in the presence of people.
- It is associated with: *anxiety, palpitaion & hyperventilation.*
- CT & MRI: normal.

CAUSES OF TRANSIENT HEM1PLEGIA

1. **T**ransient ischemic attacks (TIAs).
2. **T**odd's paralysis (post-epileptic).
3. Demyelinating disease (DS).
4. **H**emiplegic migraine.
5. **H**ysterical.

BLOOD SUPPLY OF THE BRAIN

The blood reaches the brain through **two** Systems of blood vessels:

I. THE CAROTID SYSTEM.

II. THE VERTEBROBASILAR SYSTEM.

I. THE CAROTID SYSTEM

INTERNAL CAROTID ARTERY — ANATOMY

Each internal carotid artery enters the cranial cavity through the:
Carotid foramen and Canal to the Cavernous sinus where it lies lateral to the optic Chiasma.

The artery in the sinus gives off 3 SMALL BRANCHES:

- The ophthalmic artery.
- The anterior choroidal artery.
- The posterior communicating artery.

The artery finally divides into its 2 MAIN TERMINAL BRANCHES:

The ***middle cerebral artery*** and the ***anterior cerebral artery***.

INTERNAL CAROTID ARTERY — OCCLUSION

- **Etiology:** most commonly *atherosclerosis* of the artery.
- **Age:** most commonly the *4th -6th decades*.
- **Sex:** most commonly affecting the *males*.

- **Clinical Picture:**

I. Recurrent Carotid TIAs: "may preceed complete occlusion"

1. Transient sudden Headache.
2. Transient Convulsions.
3. Transient Ipsilateral Blindness.
4. Transient Contralateral Hemiparesis & Hemihyposthesia.
5. Transient Aphasia in lesions of the DOMINANT HEMISPHERE.

" These manifestations indicate insufficiency of the Internal Carotid Artery, (Carotid TIAs) which may terminate in complete occlusion of the artery."

II. STROKE (Complete Carotid Occlusion): "may be preceeded by TIAs"

1. Ipsilateral Blindness.
2. Contralateral Hemiplegia & Hemihyposthesia.
3. Aphasia in lesions of the DOMINANT HEMISPHERE.
4. Contralateral Homonymous Hemianopia.

1. THE MIDDLE CEREBRAL ARTERY

It runs on the lateral surface of the cerebral hemisphere.
It supplies the **lateral aspect of the anterior 3/5s of the cerebral of hemisphere**.
It gives the following branches:

1. CAPSULAR BRANCH
2. CORTICAL BRANCHES.

1. CAPSULAR BRANCH (Lenticulo-striate artery):

ANATOMY

- It supplies all the dorsal half of the IC.

CAPSULAR BRANCH (Lenticulo-striate artery):

OCCLUSION

- It is the most commonly occluded artery resulting in:
  1. Contralateral complete hemiplegia affecting the UL & LL to the same extent.
  2. Contralateral UMNL of the facial & hypoglossal nerves.
  3. Contralateral hemihyposthesia of the cortical type.
  4. Contralateral hemianopia may occur.
  5. No **coma**, No **convulsions**, No **aphasia**.

2. CORTICAL BRANCHES:

ANATOMY

Frontal branch
- *Motor area of face & UL*
- *Motor speech area (44)*
- *Motor writing area (45)*

Parietal branch
- *Sensory area of UL*
- *Angular gyrus (39)*
- *Supramarginal gyrus (37)*
- *Upper fibres of optic radiation*

Temporal branch
- *Auditory areas*
- *Lower fibres of optic radiation*

CORTICAL BRANCHES:

OCCLUSION

Frontal branch
Contralateral Faciobrachial monoplegia
Motor aphasia (in lesions of Domin Hemi)
Agraphia (in lesions of Domin Hemi)

Parietal branch
Cortical sensory loss in UL
Alexia (in lesions of Domin Hemi)
Apraxia of both sides (in lesions of Domin Hemi)
Contralateral Lower quadrantic Homo Hemianopia

Temporal branch
Auditory agnosia (in lesions of Domin Hemi)
Contralateral Upper quadrantic Homo Hemianopia

MAIN ARTERY OCCLUSION

1. Coma & may be Convulsions at the onset *(because the lesion is extensive).*
2. Contralateral hemiplegia affecting UL more than LL.
3. Contralateral hemihyposthesia with more cortical sensory loss in UL.
4. Contralateral homonymous hemianopia.
5. APHASIA *(Motor aphasia, Agraphia, Alexia, Auditory agnosia)* and APRAXIA *(in lesions of Domin Hemi).*

2. THE ANTERIOR CEREBRAL ARTERY

It runs on the medial surface of the cerebral hemisphere.
It supplies the **medial aspect of the anterior 3/5s of the cerebral of hemisphere**.
It gives the following branches:

1. CAPSULAR BRANCH
2. CORTICAL BRANCHES.

1. **CAPSULAR** BRANCH (Heubner's artery):
   - It supplies the ventral half of the anterior limb of the IC.

ANATOMY

CAPSULAR BRANCH (Heubner's artery):
- Contralateral Facio-brachial monoplegia.
  (proximal more than distal)

OCCLUSION

2. CORTICAL BRANCHES:

ANATOMY

Frontal branch

Prefrontal area

- *Mentality*
- *Personality*
- *Inhibition of primitive reflexes*

Paracentral branch

- *Motor area of LL*
- *Sensory area of LL*
- *Paracentral lobule*

Callosal branch

- *Corpus callosum*

CORTICAL BRANCHES:

OCCLUSION

Frontal branch

Prefrontal area

Mentality changes
Personality changes
Positive grasp reflex

Paracentral branch

Contralateral Monoplegia in LL
Contralateral Cortical sensory loss in LL
Incontinence of urine (in bilateral lesions)

Callosal branch

Apraxia of the same side of the domin hemi

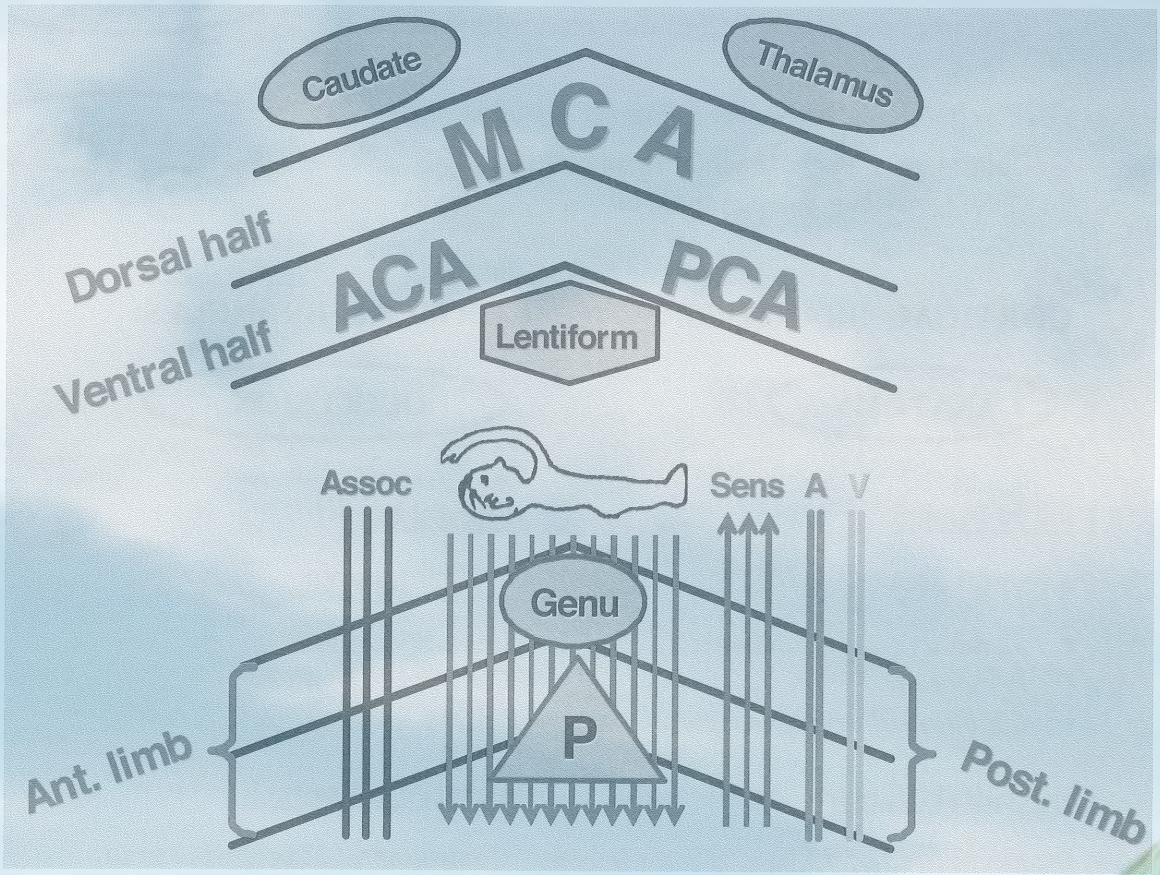
MAIN ARTERY OCCLUSION

1. Contralateral hemiplegia affecting LL more than UL.
2. Contralateral cortical sensory loss in LL.
3. Mentality & Personality changes & Positive grasp reflex.
4. Incontinence of urine *(in bilateral lesions)*.
5. Apraxia of the same side of the domin hemi.

INTERNAL CAPSULE

- **It is a broad band of white fibres lying in the depth of the cerebral hemisphere.**

- **It is formed of:**
  1. *The anterior limb:* placed between the caudate nucleus and the lentiform nucleus .
  2. *The genu.*
  3. *The posterior limb:* placed between the thalamus and the lentiform nucleus .



Blood Supply of the Internal Capsule:

1. **The dorsal half** of the anterior limb, knee, and posterior limb of the internal capsule **is** supplied by the Capsular branch of the middle cerebral artery ***(Lenticulo striate artery).***
2. **The ventral half** of the anterior limb **is** supplied by the capsular branches of the anterior cerebral artery ***(Heubner's artery).***
3. **The ventral half** of the knee and posterior limb **is** supplied by the capsular branches of the posterior cerebral artery ***(Thalamo-geniculate artery).***

Fibres passing through the Internal Capsule:

1. Pyramidal fibres descend in the genu, adjacent part of AL and anterior 1/2 of PL.
   The fibres supplying the arm are followed by those supplying the head, trunk, and lastly the LL.
2. Sensory fibres from the thalamus ascend in the PL.
3. Auditory and optic radiations pass in the posterior part of PL.
4. Associative fibres pass in the anterior part of AL.

II. VERTEBRO-BASILAR SYSTEM

ANATOMY

- Each **VERTEBRAL ARTERY** passes upwards through the vertebral foramina to enter the cranial cavity through the foramen magnum and runs upwards on each side of the medulla.
- Both vertebral arteries meet at the lower border of the pons to form one midline single artery: the **BASILAR ARTERY**.
- The basilar artery runs upwards on the ventral surface of the pons.
  - It gives: Small branches known as the: paramedian arteries to the brain stem.
  - It then divides: into its two terminal branches the: posterior cerebral arteries.

- Each POSTERIOR CEREBRAL ARTERY supplies the following *lobes:*
  - The whole occipital lobe.
  - The posterior part of the temporal lobe.

  } **posterior *2/5s* of the cerebral hemisphere**

- Each POSTERIOR CEREBRAL ARTERY gives the following *branches:*
  1. Posterior **Communicating** artery.
  2. **Capsular** branch supplying the ventral half of the posterior limb of the internal capsule, the thalamus and the geniculate bodies (Thalamo-geniculate artery).
  3. **Cortical** branches to the posterior *2/5s* of the cerebral hemisphere.

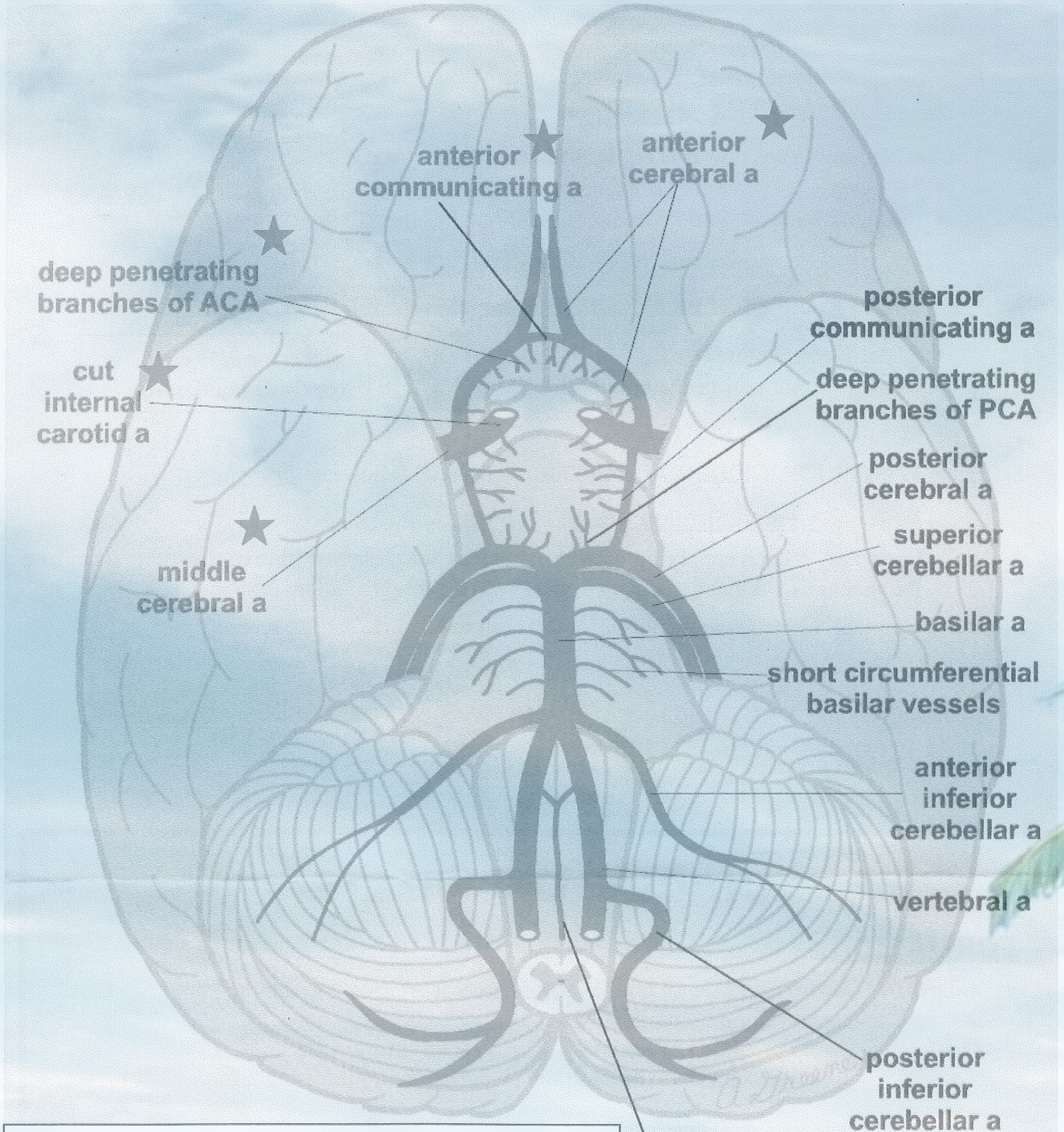
- In its course, the VERTEBRO – BASILAR system gives the following *branches*:
  - Two **spinal arteries** which unite to form the anterior spinal artery.
  - Three **cerebellar arteries** on each side: *Superior*, *Middle* & *Inferior*.

CIRCLE OF WILLIS

- **FORMATION:**
  1. *Anteriorly:* by the union of both anterior cerebral arteries through the anterior communicating artery.
  2. *Posteriorly:* by the union of each posterior cerebral artery with the ICA of the same side through the posterior communicating artery.

  Therefore, in the circle of Willis:
  - The two carotid arteries communicate with each other, and with the vertebro-basilar system.
- **SITE:** It is present in the subarachnoid space at the base of the brain.
- **RELATIONS**
  - *Anteriorly:* to the olfactory tract & optic chiasma.
  - *In the middle:* to the cavernous sinus & optic chiasma.
  - *Posteriorly:* to the midbrain & oculomotor nerve.

CIRCLE OF WILLIS



With ★ = Carotid System

Without ★ = Vertebro – Basilar System

II. VERTEBRO-BASILAR SYSTEM — OCCLUSION

VERTEBRO-BASILAR ARTERY (MAIN VESSEL) OCCLUSION

a) Recurrent Vertebro-Basilar TIAs: "may preceed complete occlusion"

1. Syncope.
2. Diplopia.
3. Ophthalmoplegia.
4. Vertigo or tinnitus.
5. Bulbar symptoms (dysphagia, dysarthria, nasal regurgitation, and hoarseness of voice).
6. Hemiparesis, hemianaesthesia or parasthesias.
7. Ataxia.

"These manifestations indicate insufficiency of the Vertebro-Basilar Artery, (Vertebro-Basilar TIAs) which may terminate in complete occlusion of the artery."

b) STROKE (Complete occlusion): "may be preceeded by TIAs"

- Deep coma.
- Complete quadriplegia with decerebrate rigidity.
- Bulbar paralysis.
- Hypoventilation → acute respiratory failure.

} **Usually fatal**

POSTERIOR INFERIOR CEREBELLAR ARTERY (PICA) — ANATOMY

- It arises as a branch of the vertebral artery & terminates to supply the inferior part of cerebellum.
- **It runs upwards on the lateral aspect of the medulla & supplies its upper lateral part which contains:**
  1. Some of the reticular formation nuclei.
  2. Part of the vestibular nucleus.
  3. Sympathetic fibres to the eye and face.
  4. The nuclei of 9^{th}, 10^{th} & 11^{th} cranial nerves.
  5. Spinal sensory nucleus of the 5th nerve for pain & temp sens of the same side of the face.
  6. Spinal lemniscus carrying pain & temp sens from the opposite side of the body.

POSTERIOR INFERIOR CEREBELLAR ARTERY (PICA) — OCCLUSION

1. Acute onset associated with syncope, hiccup, vomiting, vertigo and pain over the face.
2. Ipsilateral cerebellar ataxia (nystagmus, dysarthria, incoordination).
3. Ipsilateral Horner's syndrome.
4. Ipsilateral palato-pharyngeo-laryngeal paralysis & weakness of sternomastoid & trapezius.
5. Ipsilateral loss of pain and temperature sensations over the face.
6. Contralateral loss of pain and temperature sensations over the body.

BRAIN STEM BRANCHES OCCLUSION

"Syndromes of Brain Stem Hemiplegia: Crossed Hemiplegia"

POSTERIOR CEREBRAL ARTERY OCCLUSION

1. CAPSULAR BRANCH (Thalamo-geniculate artery): ANATOMY

- It supplies: the ventral half of the posterior limb of the internal capsule, the thalamus and the geniculate bodies.

CAPSULAR BRANCH (Thalamo-geniculate artery): OCCLUSION

A) Thalamic Syndrome:

1. Thalamic pain: severe burning pain on the contralateral side.
2. Reflex dystrophy of the shoulder girdle & arm secondary to the pain.
3. Contralateral hemianaesthesia.
4. Extrapyramidal manifestations: due to ischemia of the basal ganglia.

B) LL monoplegia.

2. CORTICAL BRANCH: ANATOMY

- It supplies: the posterior *2/5s* of the cerebral hemisphere.

CORTICAL BRANCH: OCCLUSION

1. Contralateral homonymous hemianopia + preserved light reflex + preserved macular vision.
2. Visual agnosia *(in lesions of Domin Hemi).*

MAIN ARTERY OCCLUSION

1. Thalamic Syndrome.
2. Contralateral homonymous hemianopia + preserved light reflex + preserved macular vision.
3. Visual agnosia *(in lesions of Domin Hemi).*

NEUROGENIC BLADDER

I. LMNL *(Lesions at the level of the reflex arc)*

1. Sensory Atonic Bladder (Lesion in the afferent sensory fibres)
   - Absence of the sense of fullness of the bladder.
   - Retention of urine with overflow.
2. Motor Atonic Bladder (Lesion in the efferent motor fibres)
   - Preservation of the sense of fullness of the bladder.
   - Retention of urine due to inability to evacuate the bladder voluntarily.
   - Catheterisation is needed.
3. Autonomic Bladder (Lesion in spinal center $S_{2,3,4}$ or in both afferent & efferent fibres)
   - Involuntary & Incomplete evacuation of the bladder .
   - Evacuation of the bladder occurs by its myogenic contraction.

II. UMNL *(Lesions above the level of the reflex arc)*

1. Acute:
   - Retention of urine with overflow.
2. Gradual:
   - Partial lesion: Precipitancy of micturition.
   - Complete lesion: Automatic Bladder:
     - *Complete evacuation of the bladder.*
     - *Evacuation of the bladder occurs by the spinal reflex arc.*

For any disturbance in bladder function to occur, the lesion should be bilateral.

I. UMNL

1. **Acute:**
   - Retention with overflow.
2. **Gradual:**
   a. Partial: Precipitancy.
   b. Complete: Automatic bladder.

II. LMNL

1. Sensory atonic bladder.
2. Motor atonic bladder.
3. Autonomic bladder.

PARAPLEGIA

DEFINITION

Paralysis or Paraparesis of both lower limbs.

TYPES

- SPASTIC PARAPLEGIA: due to UMNL (any lesion in the Pyramidal pathway).
- FLACCID PARAPLEGIA: due to LMNL (any lesion from the AHCs till the muscles).

SPASTIC PARAPLEGIA

DEFINITION

- Paralysis or paresis of both LLs due to BILATERAL PYRAMIDAL tract lesion.

TYPES

I. SPINAL PARAPLEGIA *The most common type*

1. **Focal:** paraplegia **with a level**
2. **Systemic** } paraplegia **without a level**
3. **Disseminated** }

II. CEREBRAL PARAPLEGIA *The least common type*

A. Causes in the Parasagittal Region: (area of cortical presentation of LLs)

1. Traumatic e.g. Depressed fracture of skull, Subdural hematoma.
2. Vascular e.g. Superior Sagittal Sinus thrombosis.
3. Inflammatory e.g. Encephalitis, Meningio-encephalitis.
4. Neoplastic e.g. Parasagittal meningioma.
5. Degenerative e.g. Cerebral palsy (Little's disease).

B. Causes in the Brain Stem:

- Syringobulbia.
- Midline brain stem tumours.

CAUSES OF SPINAL PARAPLEGIA

1. FOCAL PARAPLEGIA: *due to:*

A. Compression: *Acute, Subacute, Chronic*

1. Acute (extramedullary)

- TB of the spine: Pott's disease. } Inflammatory
- Fracture or fracture-dislocation of the vertebrae. } Traumatic
- Disc prolapse.

2. Subacute (extramedullary) or (intramedullary)

- TB of the spine: Pott's disease. } Inflammatory
- Tumours of the spine. } Neoplastic

TUMOURS THAT COMPRESS THE SPINAL CORD MAY ARISE FROM:

1. *Extramedullary:* (Outside the spinal cord: Vertebrae, Meninges, Roots)
   - Vertebrae: *Extradural:* e.g. **1ry** osteosarcoma, or **2ry** deposits.
   - Meninges: *Dural* e.g. meningioma.
   - Roots: *Intradural* e.g. neurofibroma.
2. *Intramedullary:* (Inside the spinal cord parenchyma itself)
   - Glioma or Ependymoma.

3. Chronic (intramedullary) or (extramedullary)

- Glioma or Ependymoma. } Neoplastic
- Syringomyelia. } Cavitary
- Spondylosis. } Degenerative

B. Inflammatory:

- Transverse myelitis.

C. Vascular:

- Anterior spinal artery occlusion.

2. SYSTEMIC PARAPLEGIA: *due to:*

- A systemic disease affects one or more systems selectively & is usually bilateral & symmetrical.
- A systemic disease affects the pyramidal tracts, either alone or with other tracts → paraplegia.

A. Heridofamilial:

1. Hereditary spastic paraplegia.
2. Hereditary ataxias, e.g. *Friedreich's ataxia* or *Marie's ataxia*.

B. Secondary:

1. Pellagral lateral sclerosis.
2. Subacute combined degeneration (SCD).

C. Idiopathic:

- Motor neurone disease (MND).

3. DISSEMINATED PARAPLEGIA: *due to:*

- ***A disseminated disease is a multifocal disease of the same nature:***
  1. Disseminated Sclerosis (DS): " Refer to DS ".
  2. Disseminated Encephalo-myelitis (DEM).

DIAGNOSIS OF SPASTIC PARAPLEGIA

I. CLINICAL DIAGNOSIS

Criteria to diagnose Spastic Paraplegia

1. Paralysis or paresis of both LLs.
2. Hypertonia (clasp knife spasticity).
3. Hyper-reflexia (and may be clonus).
4. Positive Babinski sign.
5. Sphincteric troubles, especially *precipitancy of micturition*.

II. ANATOMICAL DIAGNOSIS

1 *Is it: Systemic, Disseminated or Focal paraplegia ??*

- **Systemic:** **Paraplegia without a level**
  - *Onset:* Gradual *Course:* Progressive.
  - *Affection:* Symmetrical affection.
- **Disseminated:** **Paraplegia without a level**
  - *Onset:* Acute *Course:* Remissions & exacerbations.
  - *Affection:* Multiple nervous system affection (dissemination).
- **Focal:** **Paraplegia with a level**
  - Below the level: UMN manifestations & Sensory loss.
  - At the level: LMN manifestations & Radicular manifestations.

2 *Clinical Picture of Focal Paraplegia:*

1. Vertebral manifestations: "in vertebral causes only"

- Localised pain & tenderness.
- Localised swelling & deformity.

2. Radicular manifestations: "in extra-medullary causes only"

a) Posterior root affection:
- Early: Girdle pain: back pain referred to the distribution of the affected root.
- Late: Sensory loss: in the dermatome supplied by the affected root.

b) Anterior root affection:
- Localised LMN weakness in the muscles supplied by the affected root.

3. Cord manifestations:

a- Sensory affection:

Extra-medullary lesions

Sensory level: below which all types of sensations are diminished.
Sacral affection: early loss of sensations in the saddle area ($S_{3,4,5}$).

Intra-medullary lesions

Jacket sensory loss: of dissociated nature (lost pain & temp. & preserved touch & deep).
Sacral spare: preserved sensations in the saddle area ($S_{3,4,5}$).

b- Motor affection:
- LMNL (with fasciculations): at the level of the lesion due to affection of the AHCs.
- UMNL: below the level of the lesion due to affection of the descending pyramidal tracts.

c- Sphincteric affection: *Early in Intra-medullary & Late in Extra-medullary lesions*
- Acute lesions: Retention with overflow.
- Gradual lesions: Precipitancy (partial lesion), then: Automatic bladder (complete lesion).

3 *Is it: Extra-medullary or Intra-medullary paraplegia ??*

- Refer to the table.

III. DIAGNOSIS OF STAGE OF PARAPLEGIA

1. Stage of flaccid paralysis (shock stage)

- It occurs in acute lesions & lasts from 2-6 weeks.
- It is characterized by:
  - *Paralysis of both LLs.*
  - *Hypotonia & Hyporeflexia.*
  - *No plantar response.*
  - *Retention of urine with overflow.*

2. Stage of spastic paralysis (established paraplegia)

- It occurs in: **a)** Recovery of acute lesions, or **b)** Gradual lesions from the start.
- It passes into **2 stages:**

A. Paraplegia in Extension:

- It occurs in INCOMPLETE injury of the spinal cord, where there is: affection of the pyramidal & sparing of the extra-pyramidal tracts.
- It is characterized by:
  - Hypertonia (spasticity): affects the **extensors** more than the flexors.
  - Deep reflexes: exaggerated & may be CLONUS.
  - PRECIPITANCY of micturition.

B. Paraplegia in Flexion:

- It occurs in COMPLETE transection of the spinal cord, where there is: affection of both pyramidal & extra-pyramidal tracts.
- It is characterized by:
  - Hypertonia (spasticity): affects the **flexors** more than the extensors.
  - Deep reflexes: less exaggerated & NO CLONUS.
  - AUTOMATIC BLADDER.
  - Positive mass reflex:

    Any stimulus below the level of the lesion, results in: withdrawal of the legs, sweating below the level, micturition, defecation, & ejaculation.

	Paraplegia in extension	Paraplegia in flexion
1. Cause	Pyramidal lesion	Pyramidal & extrapyramidal
2. Hyypertonia	More in extensors	More in flexors
3. Position of LLs	Extended	Flexed
4. Deep reflexes	Exaggerated	Less exaggerated
5. Clonus	Present	Absent
6. Mass reflex	Absent	May be present
7. Bladder	Precipitancy	Automatic bladder

IV. ETIOLOGICAL DIAGNOSIS

- Differential Diagnosis of the causes.

INVESTIGATIONS

I CSF

Formation & circulation:

- Normally the CSF is a clear colourless fluid, formed in the cerebral ventricles.
- It leaves the ventricles through the lateral and medial foramina of the fourth ventricle to the subarachnoid space covering the brain & the spinal cord.
- It is then finally absorbed into the blood to internal jugular veins.

Functions:

- Shock absorption: it acts as a **cushion** for the CNS and protects it from traumatic injury.
- Nutrition: it contains sugars & other elements that are used by the CNS cells.
- Waste disposal: it removes waste products produced by metabolism of CNS cells.

Absorption of the CSF from the subarachnoid space into the blood stream takes place through the **arachnoid villi**. When the CSF pressure is greater than the venous pressure, CSF will flow into the blood stream. However, the arachnoid villi act as "**one way valves**" ...if the CSF pressure is less than the venous pressure, the arachnoid villi will NOT let blood pass into the ventricular system.

1) CSF pressure:

It is measured by tapping the CSF in the lumbar region, using a needle connected to a spinal manometer.

- Normally it is 120 – 140 mmH_2O.
- In extra-medullary compression: it is markedly diminished.
- In intra-medullary compression: it is moderately diminished.

2) Queckenstedt's test: "UNRELIABLE TEST"

- Bilateral jugular vein compression normally ↓ the flow of the CSF from the SAS to the blood & leads to rapid rise of CSF pressure in the manometer. Release of the compression leads to rapid drop of pressure to normal.
- In Extra-medullary Compression: **(complete dynamic block)**
  There is no change of CSF pressure, due to complete obstruction of the SAS.
- In Intra-medullary compression: **(partial dynamic block)**
  There is a slower & lesser rise of pressure, due to incomplete obstruction of the SAS.

3) CSF contents:

Normal CSF contents

- Proteins: 20 - 40 mg / 100 ml.
- Cells: 0 - 5 / HPF., *mainly lymphocytes*.
- Chlorides: 720 - 750 mg / 100m1.
- Sugar: 50 - 80 mg / 100 ml.

- **In Extra-medullary compression:**

  There is marked increase in proteins leading to: **" Froin's Syndrome "**

  - Spontaneous coagulation.
  - Xanthochromia (yellowish discoloration).
  - Cyto-albuminous dissociation (↑ proteins BUT near-normal cell count).

- **In Intra-medullary comression:**
  - There is no significant change.

2 CT scan & MRI

- Accurate in localization of the lesion.

3 Plain X-Ray of the Spine

" in vertebral causes only "

- Destruction of the vertebrae: *Pott's disease, Trauma or Tumours.*
- Narrowing of intervertebral spaces ± osteophytes: *Spondylosis & acute disc prolapse.*

4 Myelography

A radio-opaque substance is injected into the SAS and then X-ray pictures are taken:

- Extra-medullary compression: complete block with short tails (saddle shape).
- Intra-medullary compression: ↑ width of the cord with long tails (fusiform shape).

	Extra-medullary	Intra-medullary
HISTORY		
Onset	Painful	Painless
Sphincteric affection	Late or Absent	Early
CLINICAL PICTURE		
Sensory	***Sensory level:*** below which all types of sensations are diminished. ***Sacral affection:*** early loss of sensations in the saddle area ($S_{3,4,5}$).	***Jacket sensory loss:*** of dissociated nature (lost pain & temp. & preserved touch & deep). ***Sacral spare:*** preserved sensations in the saddle area ($S_{3,4,5}$).
Sphincteric affection	Late or Absent	Early
INVESTIGATIONS		
CSF	Marked ↓ of pressure. Complete dynamic block. Froin's syndrome.	Moderate ↓ of pressure. Partial dynamic block. No Froin's syndrome.
CT & MRI	Diagnostic	Diagnostic
Plain X-Ray Myelography	Possible vertebral lesion Saddle-shaped block	Normal Fusiform-shaped block

TREATMENT

I. GENERAL CARE

1. Care of the skin:

- Frequent change of: the patient's position (every 2 hours), and of the bed sheets.
- Frequent wash of: skin of the back & pressure points by alcohol followed by powder.

2. Care of nutrition & fluid balance:

- Tube feeding & IV fluids.

3. Care of the bladder:

- Catheterisation & urinary antiseptics.
- Parasympathomimetics: in case of retention.

4. Care of the bowel:

- Daily enema.

II. SYMPTOMATIC

1. Vitamin and tonics.
2. Muscle relaxants.

III. PHYSIOTHERAPY.

IV. SPECIFIC: " treatment of the cause "

e.g. Anti-tuberculous drugs in case of Pott's disease.

SPECIAL FORMS OF PARAPLEGIA

TRANSVERSE MYELITIS

DEFINITION

It is an ACUTE INFLAMMATION affecting the **gray & white** matter in one or more adjacent spinal cord segments; commonly the thoracic segments.

ETIOLOGY

1. **IDIOPATHIC.**
2. **INFECTION:**
   - *Viral:* HS, HZ, CMV, EBV, Influenza, Coxsakie B or Echo virus.
   - *Bacterial:* TB, Syphilis, Diphtheria.
3. **IMMUNOLOGIC:**
   - *Following vaccination.*
   - *Associated with vasculitis:* e.g. SLE.
4. **IATROGENIC & TOXIC:**
   - *Post lumbar.*
   - *IV heroin.*
5. **DEMYELINATING:** DS & DEM.

CLINICAL PICTURE

Shock stage: (2 – 6 weeks)

- Paraplegia: Flaccid paraplegia + sensory level below which all types of sensations are lost.
- Sphincteric: Retention of urine with overflow.

Recovery stage:

- Paraplegia: Spastic paraplegia + sensory level below which all types of sensations are lost.
- Sphincteric: Precipitancy of micturition.

DIFFERENTIAL DIAGNOSIS

- Acute cord compression.
- Anterior spinal artery occlusion.
- Guillain-Barré syndrome.

INVESTIGATIONS

CSF examination:

- Increased both cells and proteins.
- In DS there is high immunoglobulin level.

MRI:

- To exclude acute cord compression.

TREATMENT

1. Nonspecific symptomatic ttt.
2. CORTICOSTEROIDS:
   - Prednisone (1 mg / Kg / day, orally): in: *Idiopathic cases, SLE, DS.*
   - ACTH (IM): in: *DS.*

ANTERIOR SPINAL ARTERY OCCLUSION

ETIOLOGY

Thrombosis, Embolism.

CLINICAL PICTURE

Similar to Transverse Myelitis but there is sparing of the deep sensations because the posterior columns of the spinal cord are supplied by the posterior spinal artery.

SYRINGOMYELIA

DEFINITION

It is a chronic disorder characterised by:

1. **Lesion:** Long CAVITIES surrounded by GLIOSIS.
2. **Site:** In the CENTRAL part of the **gray matter** of the spinal cord:
   - Most commonly: in the lower cervical & upper thoracic segments.
   - Less commonly: in the lumbar segments OR may extend up to the medulla (Syringobulbia).

ETIOLOGY

1. Congenital.
2. Idiopathic.

CLINICAL PICTURE

- Age: Commonly between 15 & 35 years.
- Onset and course: Gradual onset & slowly progressive course.

I. Cervical Syringomyelia:

1. Motor manifestations:
   - ***Early:*** In UL: Localised LMN weakness + fasciculations *due to encroachment on **AHCs***.
   - ***Late:*** In LL: Spastic paraplegia (UMN) *due to encroachment on the **pyramidal tracts***.
2. Sensory manifestations:
   - **Jacket sensory loss** of **dissociated nature** in the area of skin supplied by the affected segments with sacral spare.
3. Autonomic manifestations:
   - Morvan's syndrome.
   - Horner's syndrome.
4. Associated skeletal anomalies pes cavus, & spina bifida.

II. Lumbar Syringomyelia: Picture of: **epiconus lesion** (see later).

INVESTIGATIONS

- MRI.
- Myelography.

TREATMENT

1. X-ray irradiation of the affected region of the cord.
2. Physiotherapy & symptomatic treatment.

SYRINGOBULBIA

- It is a lesion in the MEDULLA similar to that in syringomyelia.
- The lesion may start in the medulla OR may be an upward extension of a cervical syringomyelia.
- It involves: the spinal nucleus of 5th cranial nerve Bulbar nuclei & Vestibular nucleus.

CLINICAL PICTURE

- PAIN IN THE FACE followed by loss of sensation of dissociated nature.
- BULBAR SYMPTOMS with lost palatal and pharyngeal reflexes + **wasting of the tongue**.
- VERTIGO.

CAUDA EQUINA

ANATOMY

- During intra-uterine life, the rate of growth of the vertebral column is faster than the rate of growth of the spinal cord.
- So, normally the spinal cord ends at the junction of L_1 & L_2 vertebrae.
- From this level downwards, the spinal canal is not empty; it is occupied by the collection of lumbo-sacral roots, known as the **" Cauda Equina ."**

DEFINITIONS

Cauda Equina	=	collection of lumbo-sacral ***roots*** ion the lower part of the spinal canal.
Conus Medullaris	=	the lowermost 3 segments of the ***spinal cord***: $S_{3,4,5}$.
Epiconus	=	the 4 segments of the ***spinal cord*** above the conus medullaris: $L_{4,5}$ $S_{1,2}$.

CAUDA EQUINA LESION

ETIOLOGY

- Fracture or fracture-dislocation of the lumbar vertebra.
- Disc prolapse.

} **Traumatic**

- TB of the spine: Pott's disease.

} **Inflammatory**

- Neoplastic diseases of the spine:
  - Primary: e.g. osteosarcoma.
  - Secondary (metastatic) from a primary carcinoma.

} **Neoplastic**

- Lumbar spondylosis.

} **Degenerative**

CLINICAL PICTURE

I. MOTOR MANIFESTATIONS

- Paralysis or weakness of LMN nature in the LL (in one or both LLs).
- The distribution of weakness depends on the affected roots.

The affection is usually unilateral, **BUT:** if bilateral, it is assymetrical

II. SENSORY MANIFESTATIONS

- *Early: Radicular pain:* in the distribution of the femoral or sciatic nerves.
- *Late: Radicular sensory loss:* the distribution depends on the affected roots.

The affection is usually unilateral, **BUT:** if bilateral, it is assymetrical

III. AUTONOMIC MANIFESTATIONS

1. Sphincteric manifestations:
   - They are in the form of:
     a) Sensory atonic bladder, or b) Motor atonic bladder, or c) Autonomic bladder.
2. Trophic changes.

Distribution of Motor Affection

Root	Action	Muscles
L_2	Flexion of the hip	Ileopsoas.
L_3	Extension of the Knee	Quadriceps
L_4	Dorsiflexion of the ankle	Anterior tibial group
L_5	Dorsiflexion of the toes	Anterior tibial group
S_1	Plantar flexion of the ankle & toes	Calf muscles
S_2	Flexion of the knee	Hamstrings
$S_{3,4,5}$	Anal contraction	Anal & perianal muscles

Distribution of Sensory Affection

Root	Sensory supply
L1	Upper third of the front of the thigh.
L2	Middle third of the front of the thigh.
L3	Lower third of the front of the thigh.
L4	Antero-lateral aspect of thigh, Antero-medial aspect of leg, Medial aspect of dorsum of foot, & Big toe.
L5	Lateral aspect of the thigh, Middle third of the dorsum of the foot, & Middle 3 toes.
S1	Postero-lateral aspect of the thigh & leg, Lateral third of the dorsum of the foot, & Little toe.
S2	Posterior aspect of the: Thigh & Leg & Foot.
S3,4,5	Saddle area: Anal, perianal & gluteal region.

CONUS MEDULLARIS LESION

ETIOLOGY

- Causes of Intramedullary compression.

CLINICAL PICTURE

1. Early urinary incontinence (autonomic bladder), & stool incontinence.
2. Impotence.
3. Sensory loss in the saddle area, (usually of a dissociated nature).
4. No motor or sensory affection in the LLs.

EPICONUS LESION

ETIOLOGY

- Causes of Intramedullary compression.

CLINICAL PICTURE

1. Motor:

- Paralysis or weakness in the LLs of LMN nature, in the muscles supplied by $L_{4,5}$ & $S_{1,2}$.

2. Sensory:

- Sensory loss in the dermatomes supplied by $L_{4,5}$ & $S_{1,2}$ (usually of a dissociated nature).

3. Autonomic:

- Precipitancy of urine.

FLACCID PARAPLEGIA

ETIOLOGY

1. Epiconus lesion: lesion at the AHCs of the lumbo-sacral region.
2. Cauda Equina lesion: lesion at the roots of the lumbo-sacral region.
3. Peripheral nerve lesion.
4. NMJ lesion.
5. Muscle lesion.
6. Shock stage of spastic paraplegia.

DIAGNOSIS

1. Clinical: LL paralysis of LMN nature. ***2. Anatomical.*** ***3. Etiological.***

SPONDYLOSIS

DEFINITION

- It is the gradual, progressive degeneration of the intervertebral discs, specially those which are *freely mobile* as they are more subjected to the process of wear and tear.
- The freely mobile discs are mainly found in the *cervical* and *lumbar* regions.

PREDISPOSING FACTORS

1) Old age.
2) Occupational: *repeated trauma of the spine in* **LABOURERS** *carrying excess loads.*
3) Diabetes mellitus.

PATHOLOGY

- The intervertebral disc is formed of a central gelatinous part, **"the nucleus pulposus,"** surrounded by a fibrous tissue ring, **"the annulus fibrosus".**

DISC DEGENERATION

- There is degeneration of the annulus fibrosus → herniation of the nucleus pulposus. This leads to COMPRESSION of adjacent structures.
- Since the weakest parts of the annulus fibrosus are the lateral and posterior parts, the herniation will be either: *lateral*, *posterior* or *posterolateral*.

ASSOCIATED DEGENERATION

- Sclerosis of the adjacent surfaces of the vertebrae.
- Lipping or osteophyte formation due to calcification of the prolapsed parts and ligaments.

CLINICAL PICTURE

I. CP OF CERVICAL SPONDYLOSIS

IMPORTANT SIGNS

- Hoffman sign: **reflex contraction of the thumb & index after nipping the middle finger.**
- L'hermite sign: **Electric-like sensation felt in the back & limbs on bending the neck. is due to posterior column affection in the cervical region.**

PRESENTATIONS

Any one of the 3 following manifestations depending on the direction of prolapse of the disc:

1) Features of root compression *(lateral prolapse)*

a. Anterior root compression:

- LMN paralysis or weakness in the muscles supplied by the compressed root.

Root	Action	Muscle
$C_{1,2}$	Lateral movement of neck	Sternomastoid & trapezius
$C_{3,4}$	Elevation of shoulder	Supra and infraspinatus
C_5	Abduction of shoulder	Deltoid
$C_{5,6}$	Flexion of elbow	Biceps & brachioradialis
$C_{6,7}$	**Extension of elbow**	**Triceps**
$C_{7,8}$	Extension of wrist	Extensors of wrist
C_8, T_1	Flexion of wrist & movement of small muscles of the hands	Flexors of wrist

b. Posterior root compression:

- ***Early:*** pain and paraesthesias , due to posterior root irritation, which are referred to the shoulder and ULs (brachial neuralgia).
- ***Late:*** hyposthesia in the dermatomes supplied by the compressed root.

Root	Sensory distribution
C_2	Lateral aspect of neck
$C_{3,4}$	Shoulder down to manubrium anteriorly
C_5	Lateral aspect of arm
C_6	**Lateral aspect of forearm, thenar eminence & thumb**
C_7	**Middle aspect of forearm, middle of palm, middle 3 fingers**
C_8	Medial aspect of forearm, hypothenar eminence & little finger
T_1	Medial aspect of arm

2) Features of cord compression: *(Posterior prolapse)*

a. Compression of the pyramidal tracts:

- Weakness or paralysis: with signs of UMNL below the level of compression.
- Bladder disturbances: rare & late & occur only in severe lesions.

b. Compression of the spino-thalamic tracts:

- Superficial sensory loss below the level of compression.

c. Compression of the posterior column:

- Deep sensory loss i.e. sensory ataxia below the level of compression.

3) Features of root & cord compression: *(Postero-lateral prolapse)*

a. In ULs: combined signs of LMNL in the form of wasting of muscles + signs of UMNL in the form of hypertonia and hyperreflexia, i.e. ***tonic atrophy***.

b. In LLs: weakness with signs of UMNL.

II. CP OF LUMBAR SPONDYLOSIS

- This will present with the same picture of Cauda equina lesion.

INVESTIGATIONS

1. MRI & CT scan:

- MRI is the imaging of choice.
- Will detect small disc prolapses missed by other methods of imaging.

2. Plain X-ray:

- Narrowing of the intervertebral disc spaces.
- Sclerosis of the adjacent surfaces of the vertebrae.
- Lipping or osteophyte formation.
- Straightening of the spines (loss of lordosis).

3. Myelography:

- Anterior filling defect due to disc protrusions and compression of the cord.

TREATMENT

I. Medical:

- Analgesics: e.g. NSAIDs
- Muscle relaxants.

II. Physiotherapy:

- Short wave therapy.
- Plastic neck collar: *maximum duration of* 3 months *to avoid muscle fibrosis.*
- Traction of the vertebrae.

III. Surgical: " Decompression by *laminectomy* "

INDICATIONS:

1. Failure of the medical treatment.
2. Severe intolerable pain.
3. Sensory or motor affection.
4. Bladder disturbances: rare & late & occur only in severe lesions.

SCIATICA

DEFINITION

It is radicular pain along the distribution of the sciatic nerve ($L_{4,5}$ $S_{1,2,3}$) ***i.e.*** along the back of the thigh, leg and foot.

ETIOLOGY

I. In the spinal canal: " at the lumbosacral regions "

1) Acute lumbar disc prolapse.
2) Lumbar spondylosis.
3) Fracture or dislocation.
4) Pott's disease or tumours.

II. In the intervertebral foramina:

1) Neurofibroma.
2) Radiculitis.

III. In the pelvis: " compression of sciatic plexus over the sacro-iliac joint by "

1) Malignant tumours: bladder, rectum, ovaries.
2) Pelvic abscess.
3) Pregnant retroverted uterus.

IV. In the sciatic nerve:

1) Neuritis as diabetic, alcoholic.
2) Pressure on the nerve by dislocated head of femur.
3) Wrong injection into the nerve.

V. Referred: sciatic pain secondary to hip or sacro-iliac joint disease.

The most common causes of sciatica are: • Acute disc prolapse • Lumbar spondylosis

Acute disc prolapse:

- There is sudden rupture of the annulus fibrosis followed by bulging (herniation) of the nucleus pulposus; this compresses the spinal roots.
- It may occur at any age and usually follows trauma, as lifting heavy objects.
- Plain x-ray of the back: narrowing of the intervertebral spaces with no degenerative changes.

	Acute disc prolapse	Lumbar spondylosis
Age	Any age	Middle and old age
Cause	Traumatic	Degenerative
Onset	Acute	Gradual
X-ray	- Narrowed intervertebral space	- Narrowed intervertebral space - Sclerosis, lipping & osteophytes

CLINICAL PICTURE

Symptoms

Pain and paraesthesias along the course of the sciatic nerve.

Pain is aggravated by:

1. Walking which stretches the nerve.
2. Coughing, straining or sneezing.

Pain is relieved by:

- Bed rest on a hard mattress in cases of disc lesions.

Signs

1) Sensory: Tenderness and discomfort on direct pressure on the sciatic nerve.
2) Motor: Slight LMN weakness in the muscles supplied by the nerve and the ankle reflex may be weak or lost.
3) Back signs: The paravertebral muscles are spastic; resulting in loss of lumbar lordosis, tenderness and limitation of movements of the spine.
4) Positive Kernig, Lassegue & Brudzinski signs.

INVESTIGATIONS

1) In cases with suspected lesion of the spinal canal:
   - Plain x-ray - Myelography - CT scan & MRI.
2) Rectal and vaginal examination.
3) Plain x-ray for the hip joint.
4) Urine analysis and blood sugar curve.

TREATMENT

1. Treatment of the cause.

2. In cases of disc prolapse:
   - Bed rest on a hard mattress for 2-3 weeks.
   - Analgesics (e.g. NSAIDs) and muscle relaxants to relieve the pain.
   - Physiotherapy.
   - Decompression laminectomy when there is:
     - Failure of medical treatment.
     - Frequent recurrence of pain.
     - Development of a neurological deficit (sensory or motor).

MOTOR NEURONE DISEASE

DEFINITION

- It is a systemic disease affecting the **Motor System only.**
- It may affect the: UMN, or the LMN or both.
- It is a degenerative disease of a gradual onset and progressive course.

CLINICAL PICTURE

- Age of onset: middle and old ages.
- Sex: males are more affected than females.
- Onset and Course: gradual onset and progressive course.
- Signs and symptoms:
  - They are usually bilateral (because it is a systemic disease).
  - They depend on whether the UMN the LMN or both are affected.

I. UMN AFFECTION

a) **In the spinal cord:** Syndrome of **"Lateral Sclerosis"**

Bilateral manifestations of UMNL, resulting in:

- Spastic paraplegia if the lesion is below the cervical region, or
- Spastic quadriplegia if the lesion is at the cervical region (C_1 to C_5).

b) **In the brain stem or the cerebral hemisphere:** Syndrome of **"Pseudo-bulbar palsy"**

1. Bulbar symptoms :
   - dysphagia. - nasal regurgitation.
   - dysarthria. - hoarseness of voice.
2. Quadriplegia & signs of UMNL in the upper & lower limbs.
3. Exaggerated palatal and pharyngeal reflexes.
4. Appearance of jaw reflex (if the lesion is above the pons).

II. LMN AFFECTION

The disease has a tendency to affect:

1. The AHCs: in the spinal cord, or
2. The cranial nerve nuclei: in the brain stem.

a) **In the AHCs:** Syndrome of **"Progressive muscular atrophy"**

The AHCs, mostly affected are those of the lower cervical region and to a lesser extent those of the lumbar region, resulting in weakness with signs of LMNL i.e. wasting, hypotonia, hyporeflexia and fasciculations.

b) **In the cranial nerve nuclei:** Syndrome of **"True bulbar palsy"**

1. Bulbar symptoms:
   - dysphagia. - nasal regurgitation.
   - dysarthria. - hoarseness of voice.
2. There is no quadriplegia.
3. Absent palatal and pharyngeal reflexes.
4. The tongue is wasted & shows fasciculations.

Pseudo-bulbar palsy	True bulbar palsy
1. UMNL	• LMNL
2. Associated with quadriplegia.	• No quadriplegia.
3. Exaggerated palatal & pharyngeal reflexes.	• Lost palatal and pharyngeal reflexes.
4. The jaw reflex may be exaggerated.	• Absent jaw reflex.
5. Tongue: no wasting or fasciculations.	• Tongue: small, flaccid & shows fasciculations.

III. COMBINED U.M.N. AND L.M.N AFFECTION

- Syndrome of **"Amyotrophic lateral sclerosis":**

" There are combined signs and symptoms of LMNL & UMNL ":

1. In the upper limbs there will be weakness, associated with wasting and fasciculations, (LMNL), ***plus*** hypertonia and hyperreflexia (UMNL): This is known as **TONIC ATROPHY**.
2. In the lower limbs there will be weakness with signs of UMNL.

TREATMENT

1. Vitamins: *especially vitamin E.*
2. Physiotherapy.

DD WASTING OF THE SMALL MUSCLES OF THE HAND

The small muscles of the hand are supplied by C_8 and Th_1 spinal segments. Wasting of these muscles may be due to:

1. AHC LESIONS:

1) Poliomyelitis:
   - Age: 6 months to 2 years.
   - Acute onset and regressive or stationary course.
   - The wasting is asymmetrical, affecting L.L. more than U.L.
   - No sensory changes.

2) Transverse myelitis: at C_8 and Th_1 segments.

3) Anterior spinal artery occlusion.

4) Motor neurone disease.

5) Intramedullary lesions:
   - Syringomyelia. - Lower cervical tumours.

II. ANTERIOR ROOTS AND SPINAL NERVE LESIONS: "CERVICAL AFFECTION"

1) **Cervical** spondylosis.
2) **Cervical** Pott's disease.
3) Primary and metastatic tumours of the **cervical** vertebrae.
4) Fracture and dislocation of the **cervical** vertebrae.
5) **Cervical** neurofibromatosis.

III. LOWER BRACHIAL PLEXUS LESIONS:

Thoracic outlet (inlet) syndrome: ***could be due to:***

a) Cervical rib.
b) Pancoast tumour:
c) Enlarged cervical lymph nodes.
d) Aneurysm of the subclavian artery.

IV. PERIPHERAL NERVE LESIONS:

1) All causes of mono, mononeuritis multiplex and polyneuropathy.

2) Carpal tunnel syndrome:
   - Due to compression of the median nerve in the tunnel.
   - It may be **idiopathic** or **secondary** to:
     Obesity, Amyloidosis, Myxoedema, Acromegaly & Rheumatoid arthritis.
   - The thenar muscles are mainly affected, with wasting and weakness.
   - Pain and paraesthesias especially at night associated with sensory loss over the palmar surface of the fingers.

V. MUSCLE LESIONS:

- Distal type of Gower myopathy.

VI. OTHER CAUSES:

1) Arthritis of the joints of the hand (Rheumatoid Arthritis).
2) Scleroderma & Dermatomyositis.
3) Disuse atrophy in long standing UMNL.

THE EXTRAPYRAMIDAL SYSTEM

DEFINITION

- It includes all fibres that can influence the MEP and do not pass in the pyramidal tract.

FUNCTION

1. ***REGULATION*** *of the voluntary motor activity.*
2. *Regulation of the emotional & associated movements.*
3. *Inhibition of the muscle tone.*

DYSFUNCTION

1) STATIC TREMORS **(disturbance in *REGULATION* *of the voluntary motor activity*):**

- Regular: Parkinsonism.
- Irregular: Chorea, athetosis, dystonia.

2) BRAYKINESIA **(disturbance in *REGULATION* *of the emotional & associated mov*):**

- Mask face (Immobile face with infrequent blinking…….staring look).
- Monotonus speech.
- Loss of swinging of the arms during walking.

3) Rigidity (Hypertonia) **(disturbance in inhibition of the muscle tone).**

PARKINSONISM (Shaking Palsy)

DEFINITION

- It is a condition in which there are ***Static tremors (regular)*** associated with:
  Bradykinesia & ***Rigidity (hypertonia)*** of the muscles of the body.

ETIOLOGY

Deficiency of Dopamine in the Basal Ganglia

I. Idiopathic: (paralysis agitans = unknown cause)

- Age: Above 50 years.
- Sex: Both sexes are equally affected.

II. Secondary:

1. INFLAMMATORY: ENCEPHALITIS.
2. VASCULAR: CEREBRAL ATHEROSCLEROSIS.
3. Toxic: Major tranquilizers *(Phenothiazines)*.
4. Traumatic: Repeated trauma to the head as in boxers.
5. Tumours: of the basal ganglia.

CLINICAL PICTURE

1. Static Tremors:

- Regular, and occur at the rate of: 4 – 8 / second.
- They give the hand the ***pill-rolling appearance.***

These tremors increase with:	emotional stress & fatigue.
These tremors decrease with:	sleep and during active voluntary movements.

2. Bradykinesia: Loss of emotional and associative movements

- Mask face (Immobile face with infrequent blinking…….staring look).
- Monotonus speech.
- Loss of swinging of the arms during walking.

3. Rigidity: Hypertonia

A) Distribution:
- Proximal more than the distal muscles.
- Flexors of the neck, trunk & limbs resulting in the: ***gorilla-like attitude.***

B) Character:
- Lead pipe: present throughout the act to the same degree, or:
- Cog wheel: interrupted by the tremors.

C) Gait:
- Short steppage: slow shuffling (Difficulty in starting the act of walking).

	Rigidity	Spasticity
Site of lesion	- Extra-pyramidal.	- Pyramidal.
Distribution	- Proximal more than distal. - Flexors of ULs Flexors of LLs	- Distal more than proximal. - Flexors of ULs, Extensors of LLs.
Character	- Lead pipe or cog wheel.	- Clasp knife
Deep reflexes	- Normoreflexia or Hyporeflexia.	- Hyperreflexia.

The most common causes of Parkinsonism are:

1) Paralysis agitans. 2) Post-encephalitic. 3) Atherosclerosis.

Features	Paralysis agitans	Post- encephalitic	Atherosclerosis
Age	Above 50 years	Any age	Above 50 years
Onset	Gradual	Usually acute	Gradual
Course	Progressive	Regressive or stationary	Remissions & exacerbations
Rigidity	Tremors more than rigidity	Rigidity & tremors equally marked	Rigidity more than tremors
Possible associated features		Diabetes insipidus	Hypertension Coronary heart disease Diabetes mellitus

TREATMENT

I. Medical

- The manifestations of Parkinsonism are due to an imbalance between the levels of: ***acetylcholine*** & ***dopamine*** in the basal ganglia.
- Reduction of dopamine and relative increase in acetylcholine levels are found in all cases of Parkinsonism, whatever the cause.
- Therefore medical treatment aims to restore the balance by:
  - Decreasing acetylcholine or:
  - Increasing dopamine levels.

1) Anticholineraic drugs:

a. **Natural Belladonna alkaloids** are rarely used nowadays. They include:
- Atropine sulphate. - Hyoscine.

b. **Synthetic Belladonna-like alkaloids:**
* Cogentin 2 mg tab.
* Artane 2 and 5 mg tab.
* Parkinol 2 and 5 mg tab.

Dose: 1-2 tab. t.d.s., according to the severity of the case.
Side Effects: blurring of vision, dryness of mouth, retention of urine.
Toxic Effects: confusion and hallucinations.

Anticholinergics are best indicate in: TREMORS

2) Levo-Dopa: (Dopamine precursor):

- As dopamine does not cross the BBB, its precursor levo-dopa, which can cross it, is used instead.

Dose: 1/2 g orally daily; increase it by 1/2 g every 3 days. Max. dose: 4-6 g daily.

Side Effects:
1) Psychiatric: depression, confusion.
2) Cardiovascular: palpitations, arrhythmias.
3) Gastrointestinal: nausea, vomiting.
4) Neurological: chorea, hypotonia, epilepsy.

3) Levo-Dopa + Carbi-Dopa: (Sinemet)

- It was found that L-dopa is to some extent decarboxylated in the liver to dopamine before crossing the BBB resulting in:
  - The appearance of side effects due to the peripheral action of dopamine.
  - The necessity of using large doses of L-dope to insure the passage of an effective dose across the BBB.
- These disadvantages are reduced by the addition of **Carbi-dopa** which inhibits the extracerebral decarboxylation of L-dopa to dopamine.
- Carbi-dopa itself does not cross the BBB.

Dose: One tab. of Sinemet contains 250 mg L-dopa + 25 mg Carbi-dopa.
Start with one tab. daily & increase the dose by 1/2 tab. every 3 days till the case improves.

Sinemet is best indicate in: BRADYKINESIA & RIGIDITY

4) Dopamine agonists:

- They mimic the action of dopamine at receptor sites:
  - Trivastal: 20 mg t.d.s in conjunction with Sinemet.
  - Bromocriptine: 2.5 mg daily in conjunction with Sinemet.

5) Amantadine hydrochloride:

- It prevents the uptake of dopamine by the neurones: 100 mg tab. t.d.s.

6) Muscle relaxants: to minimize rigidity.

II. Surgical *In severe cases*

1) Pallidectomy.
2) Thalamotomy.

CHOREA

DEFINITION

Involuntary STATIC movements of any part of the body:

- Irregular.
- Sudden & Jerky.
- Pseudopurposive.

ETIOLOGY

I. Herido-familial: Huntington's chorea.

II. Secondary:

1. Autoimmune:	Rheumatic chorea.
2. Infective:	Post-encephalitic chorea.
3. Vascular:	Hemiballismus.
4. Toxic:	Chorea gravidarum.
5. Neoplastic:	Tumours of the Basal Ganglia.
6. Metabolic:	Hepatolenticular degeneration (Wilson's Disease).
7. Idiopathic:	Senile chorea.

RHEUMATIC CHOREA (Sydenham's Chorea)

- It is one of the major criteria of rheumatic fever.
- It may be associated with other rheumatic manifestations, but never with rheumatic arthritis.
- Age: 5-15 years.
- Sex: females more than males.

CLINICAL PICTURE

1. Choreic movements:

a) Affect the proximal muscles more than the distal muscles:

- When the patient is asked to keep his tongue protruded and unsupported by his teeth, he is unable to do so, and quickly retracts it.
- Grimacing, jerking of the shoulders, shaking of the hands and feet.

b) They increase with emotional stress and disappear during sleep.

2. Hypotonia:

- Choreic hand (scaphoid or boat-shaped hand):

  When the patient stretches his arms there is flexion at the wrist and overextension at the metacarpophalangeal and interphalangeal joints with fanning of the fingers.

3. Emotional instability:

- Sudden laughter or sudden crying.

TREATMENT

1. **Classic treatment of Rheumatic fever:**
   - Complete rest in bed.
   - Corticosteroids as prednisone: 1 mg / Kg / day.
   - Acetyl salicylic acid: 6-8 gm daily.
2. **Specific treatment of Rheumatic chorea:**
   - Reserpine : 1 mg t.d.s.
   - Haloperidol: 3 mg t.d.s.
   - Tranquilizers.

THE CEREBELLUM

The cerebellum can be divided into 3 main parts:

1. **Archicerebellum:**
   - **Function:**
     - Maintenance of Equilibrium.
   - **Lesion:**
     - Disturbance of Equilibrium.
2. **Neocerebellum:**
   - **Function:**
     - Coordination of the voluntary motor activity.
   - **Lesion:**
     - Incoordination of the voluntary motor activity.
3. **Paleocerebellum:**
   - **Function:**
     - Maintenance of the muscle tone.
   - **Lesion:**
     - Hypotonia.

ATAXIAS

DEFINITION

- Incoordination of voluntary motor activity with or without disequilibration in the absence of motor weakness.

TYPES

1. Cerebellar ataxia. 2. Sensory ataxia. 3. Vestibular ataxia. 4. Hysterical ataxia.

CEREBELLAR ATAXIA

ETIOLOGY

I. **Herido-familial:**

1. Friedreich's ataxia. 2. Marie's ataxia.

II. **Secondary:**

1. Infective: - Encephalitis - Meningitis - Cerebellar abscess.
2. Vascular: - Superior, middle and inferior cerebellar artery occlusion.
3. Toxic: - Alcohol - Antiepileptics.
4. Neoplastic. - Paramalignant syndrome.
5. Demyelinating: - DS.

CLINICAL PICTURE

1. **Incoordination** of movements of different muscles in the form of:
   1. *The eye:* Nystagmus.
   2. *The tongue:* Dysarthria in the form of staccato speech.
   3. *The head:* Nodding of the head.
   4. *The trunk:* Titubation of the trunk (swinging from side to side).
   5. *The limbs:* Intention kinetic tremors in the extremities.
2. **Hypotonia and Hyporeflexia** of the affected muscles.
3. **Gait disturbance:**
   - In archicerebellar lesions: Drunken gait (wide-base gait).
   - In neocerebellar lesions:
     - In Unilateral lesions: Deviation of the body towards the affected side.
     - In Bilateral lesions: Zigzag gait.
4. **Positive tests:** used by the neurologist to detect cerebellar ataxia.

" HERIDO-FAMILIAL ATAXIAS "

I. Friedreich's Ataxia

1. **Age:** it occurs in the 1st decade of life.
2. **Onset & course:** gradual onset and slowly progressive course.
3. **Pathology:** there is degeneration of:
   - Cerebellum especially the: ***archicerebellum***.
   - Pyramidal tracts.
   - Posterior columns.
   - Peripheral nerves.
4. **Associations:** DCM, congenital heart disease, pes cavus.

II. Marie's Ataxia

1. **Age:** it occurs in the 2nd and 3rd decades of life.
2. **Onset & corse:** gradual onset and slowly progressive course.
3. **Pathology:** there is degeneration of:
   - Cerebellum especially the: ***neo-cerebellum***.
   - Pyramidal tracts.
4. **Associations:** Mental impairment.

SENSORY ATAXIA

DEFINITION

It is ataxia due to loss of the proprioceptive (deep) sensations, at any point in their pathway:

ETIOLOGY

1. Peripheral nerve:	peripheral neuropathy.
2. Posterior root:	tabes dorsalis.
3. Posterior column:	subacute combined degeneration.
4. Medial lemniscus:	brain stem lesions.
5. Thalamus:	thalamic syndrome.

CLINICAL PICTURE

1. Kinetic tremors as tested by finger-to-nose or finger-to-finger tests appear only on **eye closure.**
2. **Rhomberg's test:** when the patient stands with his feet close together & his eyes closed, his body sways & he may fall if not supported.
3. **Stamping gait:** heavy strike of the ground on walking due to lost deep sensation.
4. **Deep sensory loss.**
5. **Hypotonia & hyporeflexia.**

PERIPHERAL NEURITIS

DEFINITION

Inflammation or degeneration of the ***peripheral nerves*** and / or the ***cranial nerves.***
Decreased conductivity of these nerves → motor, sensory & autonomic manifestations.

ANATOMICAL CLASSIFICATION

1. Mononeuropathy: affecting a single nerve in one limb.
2. Multiple Mononeuropathy: affecting 2 or more nerves in one limb.
3. Polyneuropathy: affecting many peripheral nerves in all limbs simultaneously.

PATHOLOGY

- Axonal degeneration: e.g. DM.
- Demyelination: e.g. GBS.

ETIOLOGY OF MONONEUROPATHY

1. Injury: Traumatic injection into a nerve.
2. Infection: Leprosy, Herpes zoster.
3. Invasion: Tumour.
4. COMPRESSION: Carpal tunnel syndrome.

ETIOLOGY OF POLYNEUROPATHY

I) Heridofamilial:

1. Hypertrophic interstitial polyneuropathy.
2. Peroneal muscle atrophy.
3. Freidreich's ataxia.

II) Secondary:

1. Infective:

a) Viral: mumps, measles.
b) Bacterial: diphtheria, typhoid, tetanus.
c) Mycobacterial: leprosy.

2. Immune:

- Collagen diseases as: RA, SLE, PAN & Scleroderma.
- Guillain – Barré syndrome.

3. Iatrogenic:

INH, cycloserine, phenytoin.

4. Toxic:

a) Inorganic: heavy metals *(lead, arsenic, mercury).*
b) Organic: alcohol.

5. Metabolic:

Diabetes mellitus, renal failure.

6. Endocrinal:

Acromegaly, myxoedema.

7. Neoplastic:

Bronchial carcinoma (paramalignant syndrome).

8. Nutritional: (Vitamin deficiency)

- B1: Beri-Beri.
- B6: Peripheral neuritis & dermatitis.
- B12: SCD.
- B complex especially niacin: Pellagra.

CLINICAL PICTURE OF POLYNEUROPATHY

- It will present by the 3 following manifestations in different combinations.

A. Motor:

- Weakness or paralysis of LMN nature (wasting, hypotonia, hyporeflexia. ..).
- The weakness and wasting are:
  - Bilateral and symmetrical.
  - Affecting LL: more than UL.
  - Affecting distal muscles: more than proximal muscles.
  - Affecting extensors: more than flexors.

Bilateral foot drop and wrist drop: due to weakness in the distal extensors.

- GAIT: high steppage due to the foot drop.
- REFLEXES: ankle reflex is lost, while knee reflex is preserved.
- CRANIAL NERVE AFFECTION: especially 3, 6, 7, 9, 10.

B. Sensory:

- *Early:*
  - Pain and paraesthesia in the limbs, especially distally.
- *Late:*
  - Superficial sensory impairment of the stock and glove nature.
  - Deep sensory loss especially distally.

C. Autonomic:

- Vasomotor: coldness & cyanosis of the limbs.
- Cutaneous: loss of hair & trophic ulcers.

NB: Regardless of the cause of the PN, the CP is essentially the same; variations depend on whether the motor, sensory or autonomic features predominate.

INVESTIGATIONS

1. Nerve conduction velocity tests.
2. Electromyography.

VALUES:

1. Confirm neuropathy.
2. Determine which nerves are involved.

SPECIFIC TYPES OF PN

CHARCOT - MARIE - TOOTH DISEASE

" PERONEAL MUSCLE ATROPHY "

- **Age:** A heriditary type of PN appearing during the **1st and 2nd decades**.
- **Onset & course:** Gradual onset and a very slowly progressive course.
- **Clinical picture:**

1. MOTOR MANIFESTATIONS:

The wasting & weakness start in the LLs:

- In the **peronii muscles** then the anterior tibial group, then,
- In the muscles of the **lower 1/3 of the thigh,** resulting in:

" Inverted bottle appearance "

- **Mild weakness** despite **severe wasting**.

2. SENSATIONS: are diminished especially **vibration sense**.

3. SKELETAL DEFORMITIES: are usually present e.g. **pes cavus.**

ALCOHOLIC NEUROPATHY

1. **Mainly sensory**.
2. Associated with Wernick's encephalopathy: *Amnesia + Ataxia + Ophthalmoplegia.*
3. History of alcohol intake.

LEAD NEUROPATHY

1. **Purely motor**.
2. Associated with other features of chronic lead poisoning:
   - *Anemia:* Hemolytic anemia, Sideroblastic anemia.
   - *Hypertension & lead encephalopathy.*
   - *Blue lines in the gums.*

ARSENIC NEUROPATHY

1. **Mainly sensory**.
2. Associated with other features of chronic arsenic poisoning:
   - *Hyperkeratosis of the skin.*
   - *Depigmentation of the skin..*

DIABETIC NEUROPATHY

Diabetic neuropathy is the most common complication of diabetes, both types 1 & 2.

Pathogenesis:

METABOLIC FAILURE

1. Hyperglycemia: → harmful biological changes → nerve damage.
2. Ketone bodies: → toxic to the nerves → nerve damage.
3. Polyuria: → hypovitaminosis (B_1, B_6 & B_{12}) → nerve degeneration.

VASCULAR INSUFFICIENCY

1. Diabetic microangiopathy: of the vasa nervosa.
2. Ischaemia of the nerves: due to atherosclerosis of the vasa nervosa.

Clinical picture: "MAINLY SENSORY"

1. SENSORY MANIFESTATIONS:
   - It starts as mononeuropathy & later becomes polyneuropathy.
   - *Superficial sensory affection:*
     - It starts with pain and paraesthesia followed by stock and glove hyposthesia.
   - *Deep sensory affection:*
     - It starts with ↑ muscle sense → tender calf followed by lost muscle sense.
     - There is also loss of deep reflexes and sensory ataxia.
2. MOTOR MANIFESTATIONS:
   - Minimal.
   - Late.
3. AUTONOMIC MANIFESTATIONS: "SIT"
   - **S**yncope (postural).
   - **I**mpotence.
   - **T**rophic ulcers.
   - **S**ilent myocardial infarction.
   - **I**ncontinence.

Treatment:

1. **P**roper control of diabetes.
2. **P**ain control:
   - *Analgesics.*
   - *Anti-seizure medications: Gabapentin , Carbamazepine.*
3. **P**hysiotherapy: in case of motor weakness.
4. **V**itamins B complex.
5. **V**asodilators.

DIPHTHERITIC NEUROPATHY

Neuritis is the commonest and most important nervous complication of diphtheria.

Etiology:

- Nerve damage by the exotoxin of Corynbacterium diphtheria.

Clinical Picture:

- It is **mainly motor**.
- Symptoms and signs occur 2-8 weeks after the appearance of diphtheria.
- Two main types of neuropathy may occur:
  1. *Localised type: affecting the Cranial nerves:* 3, 7, 10.
  2. *Generalised type: affecting the Peripheral nerves.*

LEPROTIC NEUROPATHY

Etiology:

Causative organism: Mycobacterium leprae.

Nerve infiltration by leprous granuloma → thickening, degeneration & ↓ conductivity.

Clinical Picture:

- The neuropathy is mainly sensory.
- The neuropathy may be of the mono-or polyneuritic types.
- Commonest nerves affected are: *lateral popliteal, ulnar, greater auricular, trigeminal & facial .*

Clinical Types:

1. **Nodular (lepromatous) leprosy:**
   - Cutaneous nodules over the face & neck which ulcerate & heal by fibrosis → **leonine face.**
   - Associated with fever and lymphoadenopathy.
2. **Maculo-anaesthetic (tuberculoid) leprosy:**
   - Patchy depigmented areas.
   - Patchy sensory loss.
3. **Mixed type.**

Treatment:

- Dapsone: 100 mg / day, orally for 2 years.
- Rifampicin: 600 mg / day, orally for 6 months.

BERI – BERI

Etiology: Vitamin B1 (Thiamin) deficiency.

Clinical Picture:

1. Dry beri-beri: peripheral sensory neuropathy.
2. Wet beri-beri: picture of high CO heart failure.
3. Wernicke's encephalopathy: *Amnesia, + Ataxia + ophthalmoplegia.*

TREATMENT:

- Thiamine 100 mg daily and vit. B complex. - Treatment of Heart failure.

PELLAGRA

Etiology:

Multiple deficiency disease due to vitamin B complex deficiency especially ***niacin***; due to:

1. Decreased intake of vitamin B complex.
2. Decreased intake of proteins of HBV (containing **nicotinic acid** or its precursor **tryptophan**).
3. Decreased absorption as in gastro-enteritis.
4. Parasitic infestation.

Clinical Picture:

I. **Cutaneous manifestations:** DERMATITIS
- Bilateral and symmetrical dermatitis followed by hyperkeratosis.
- The lesions involve the exposed areas, and over bony prominences.

II. **Gastro-intestinal manifestations:** DIARRHEA
- Mouth: stomatitis, glossitis with atrophy of the papillae (glazed tongue).
- Stomach: dyspepsia, nausea, vomiting, epigastric pain.
- Intestines: diarrhoea.

III. **Neuro-psychiatric manifestations:** DEMENTIA
- PN **mainly sensory** due to: PN degeneration.
- Para – or quadriplegia due to: Pyramidal tract degeneration (pellagral lateral sclerosis).
- Mentality changes as: dementia.

Treatment:

1. Treatment of the cause.
2. Nicotinamide 500 mg IV / day.
3. Vitamin B complex.

SUBACUTE COMBINED DEGENERATION

SCD

Etiology:

Vitamin B_{12} (cyanocobalamin) deficiency: " Refer to Hematology ".

Clinical Picture: Triad of: *Anemia + GIT + Neurological manifestations.*

I. Anaemia: (megaloblastic, refer to BLOOD).

II. Gastrointestinal manifestations:

- Refer to BLOOD.

III. Neurological manifestations:

- Combined degeneration of the peripheral nerves, posterior columns & pyramidal tracts.

1. Superficial sensory affection:

   Pains and paraesthesias in the limbs followed by stock and glove hyposthesia.

2. Deep sensory loss:

   Sensory ataxia _due to posterior column degeneration.

   Ankle reflex is lost while knee reflex is preserved due to peripheral nerve degeneration.

3. Limb weakness with + ve Babinski, BUT: with hypotonia & hyporeflexia:

   Weakness in the limbs with + ve Babinski: due to pyramidal tract degeneration

   Hypotonia & Hyporeflexia: due to peripheral nerve degeneration.

Treatment:

- Vit B_{12} (1000 µgm IM daily for 2 weeks then 100 µgm twice per week).

ACUTE INFLAMMATORY DEMYELINATING POLY – RADICUOLO – NEUROPATHY

AIDP

Guillain-Barré Syndrome

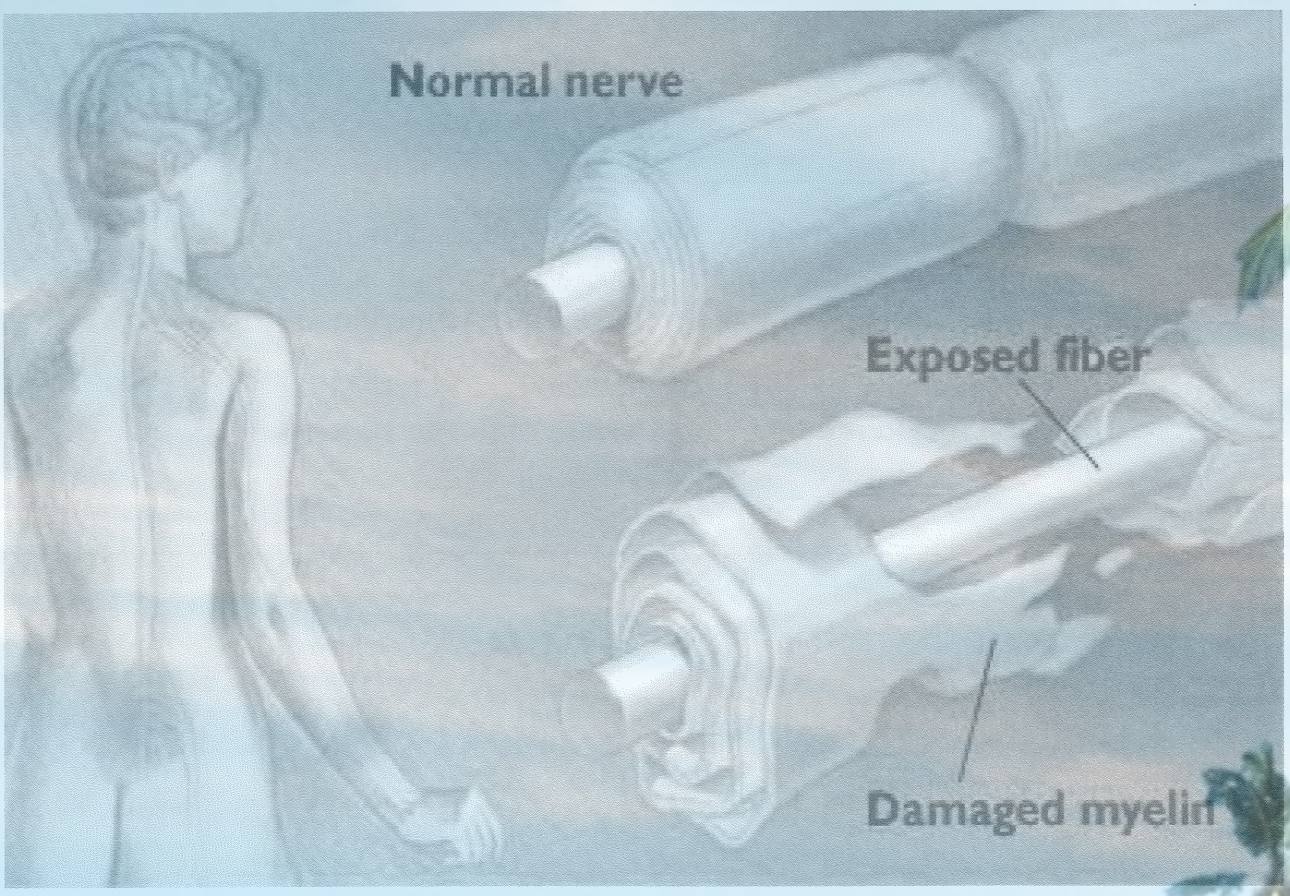
Etiology:

- It is due to an **auto-immune** disease following upper respiratory or GIT **infection**.
- Infection → antibodies against the myelin sheath of the nerves → Demyelination.

Infecting organisms

- *Bacteria:* *Campylobacter jejuni* (most common) (Gram – ve bacteria).
- Viruses: Cytomegalovirus (second most common), EBV.

Nerve damage in GBS (Demyelination)



Clinical Picture:

1. **Febrile stage:** Influenza-like attack with:
   - Fever, headache, malaise, pains all over the body, with no nervous symptoms.
2. **Latent stage:** Symptoms disappear: the patient is free for 1-3 weeks.
3. **Paralytic stage:**
   - Weakness with an ***ascending march:*** There is acute severe weakness or paralysis starting in the: LL muscles and ascending to involve the trunk and respiratory muscles, followed by the UL muscles.
   - Weakness ***proximal more than distal:*** contrary to other types of PN.
   - ***No wasting:*** In spite of the severe degree of paralysis.
   - ***Sensory impairment:*** Stock and glove hyposthesia and deep sensory loss.
   - ***Cranial nerve involvement:*** especially 7, 9, 10.

Investigations:

1. *CSF:* "**Cytoalbuminous dissociation**" (↑ proteins more than the cells).
   - During the acute phase of GBS, the characteristic findings include cyto-albuminous dissociation, which is an elevation in CSF protein without an elevation of white blood cells.
   - The increase in CSF protein is thought to reflect the widespread inflammatory disease of the nerve roots.
2. *Serology:*
   - Antibodies to glycolipids of the myelin sheath: + ve in 70 % of patients.

Prognosis:

- Prognosis is good in 85% of the cases where recovery occurs in 3-4 weeks.

Treatment:

1. **General lines:** bed rest, vitamins, physiotherapy.

2. **Specific lines:**
   - **Corticosteroids:** Prednisone , 1 mg / Kg / day.
   - **Plasmapheresis (plasma exchange):** the treatment of choice.
     "The mechanism of plasmapheresis is the removal of antibodies from the serum".
   - **IV Gamma-globulins: as effective as plasmapheresis, they inhibit antibody production.**

3. **TTT of complications:** respiratory failure: "' refer to CHEST.

CAUSES OF MOTOR NEUROPATHY

1. Peroneal muscle atrophy.
2. Lead neuropathy.
3. Diphthentic neuropathy.
4. GBS.

CAUSES OF SENSORY NEUROPATHY

1. Alcoholic neuropathy.
2. Arsenic neuropathy.
3. Diabetic neuropathy.
4. Leprotic neuropathy.
5. Vitamin deficiency neuropathy.

CAUSES OF LOST ANKLE REFLEX WITH EXAGGERATED KNEE REFLEX

1. Pellagra.
2. Subacute combined degeneration (SCD).
3. Friedreich's ataxia.

CAUSES OF LOST ANKLE REFLEX WITH PRESERVED KNEE REFLEX

1. Peripheral neuritis.
2. Epiconus.
3. Cauda equina affecting S_1 root.

CAUSES OF NERVE THICKENING

1. Hypertrophic interstitial polyneuropathy.
2. Leprosy.
3. Acromegaly.
4. Myxoedema.

MYASTHENIAS

PRIMARY MYASTHENIA (Myasthenia gravis)

DEFINITION

- It is a disorder of transmission at the neuromuscular junction, manifesting itself clinically by easy fatiguability of the skeletal muscles, specially on repetition of movement, which is relieved by rest.

ETIOLOGY

Auto-immune

1. Acetylcholine Receptor Antibodies:
   - There is production of antibodies against the acetylcholine receptors of the NMJ leading to their destruction.
   - These Acetylcholine antibodies (AChR antibodies) are found in the patient's serum.
2. Thymic Dysfunction:
   - Normally the thymus gland produces T-cell lymphocytes which participate in immune responses. Thus, thymic dysfunction may lead to disturbed immune responses with production of AChR antibodies.

CLINICAL PICTURE

- The disease affects ONLY the skeletal muscles in a DESCENDING manner.

1. Age: usually 20-40 years.
2. Sex: more in FEMALES.
3. Onset and Course: usually gradual onset and progressive course.
4. Muscles: easy **fatiguability** on repetition of movement.

5. The disease has a characteristic **descending march** affecting the following muscles:

A. ***The ocular muscles*** leading to: ptosis, diplopia & ophthalmoplegia.
B. ***The jaw muscles*** leading to: the mouth hanging open.
C. ***The facial muscles*** leading to: an expressionless appearance.
D. ***The bulbar muscles*** leading to: the tetrad of bulbar symptoms (dysphagia, dysarthria, hoarseness of voice and nasal regurgitation).
E. ***The skeletal muscles of the body:***
   - The muscles of the UL are affected before those of the LL.
   - The proximal muscles are affected more than the distal muscles.
   - The respiratory muscles are affected leading to dyspnea & repiratory failure.

6. **Diurnal variation** is noted:
   - The motor power is good in the early morning & is worst at the end of the day.

7. **Myasthenic crisis:**
   - It is characterized by aggravation of the condition & severe respiratory distress.
   - It is precipitated by : infections, operations, OR without a cause.

DIAGNOSTIC TESTS

I. CLINICAL TESTS:

a) Induction of fatigue by repetition of movement.
b) Walker's test:
   - Apply the sphygmomanometer cuff to the arm and raise the pressure above the systolic and ask the patient to do rapid repetitive movements with his hand till exhaustion occurs. Then release the cuff; if the case is myasthenia, ptosis will occur within 10 seconds.

II. PHARMACOLOGICAL TESTS:

a) Prostigmine (neostigmine) test:
   - IM Prostigmine causes improvement of fatigue after 20 minutes.

b) Tensilon test:
   - IV Tensilon causes improvement of fatigue after 2 minutes.

INVESTIGATIONS

1. **Serological tests:**
   - Acetyicholine receptor antibodies are detected in the serum of 90% of patients.

2. **EMG:**
   - Reduction of the amplitude on repeated stimulation.

3. **Radiological tests:**
   - Plain x-ray and CT of anterior mediastinum: may show *Thymus enlargement.*

4. **Muscle biopsy:**
   - Increased lymphocytes (lymphorrhages).

TREATMENT

1) Medical:

1. **Anticholinesterase drugs:**
   a) Neostigmine (prostigmine): 15 mg tds orally.
   b) Pyridostigmine (Mestinon): 60 mg tds orally.

2. **Corticosteroids:**
   - Prednisolone is used in patients not responding to anticholinesterase drugs.

3. **Immunosuppressive drugs:** e.g. *Azathioprine or cyclophosphamide:*
   - These are used in patients not responding to corticosteroids.

2) Plasmapheresis:

- This reduces the acetylcholine receptor antibody levels.

3) Irradiation or Surgical removal of the Thymus.

4) Treatment of Myasthenic crisis:

- IM Prostigmine.
- Plasmapheresis.
- Mechanical ventilation.

MYOPATHIES

DEFINITION

- A group of diseases of the skeletal muscles characterised by gradual progressive DEGENERATION of these muscles.

ETIOLOGY

I. Primary Myopathy: *"Progressive Muscular Dystrophy"*

- It is genetically determined.

II. Secondary Myopathy: *"Secondary to an underlying disease"*

1. Immune: Polymyositis & Dermatomyositis.
2. Iatrogenic: Alcohol, Corticosteroids, Statins, Zidovudine.
3. Endocrinal: Acromegaly, Cushing's syndrome.
4. Neoplastic: Bronchogenic carcinoma (paramalignant syndrome).
5. Metabolic: ↑ K or ↓ K.

PATHOPHYSIOLOGY

1. The diseased muscles in myopathy are unable to convert creatine into creatinine to get energy. *Therefore:*
   - Creatine (normally absent in urine): Will appear in urine.
   - Creatinine (normally present in urine): Will decrease in urine.
2. Deficiency of energy leads to DEGENERATION of the muscle fibres which will be replaced by fibro-fatty tissue:
   - Pseudohypertrophy: Excessive fibrofatty tissue.
   - Atrophy: Little fibrofatty tissue.

CLINICAL PICTURE *of Progressive Muscular Dystrophy*

- Age: 1st & 2nd decades.
- Onset & course: gradual onset & slowly progressive course.

- Symptoms & Signs:

1. Weakness of the skeletal muscles of the body especially those which develop early during intra-uterine life, **i.e.** the *trunk*, *shoulder* and *pelvic girdle* muscles.
2. Weakness is of LMN nature.
3. Weakness and wasting are bilateral, symmetrical and PROXIMAL more than distal, **i.e.** the shoulder and arm are more affected than the forearm and hand and the hip and thigh are more affected than the leg and foot.
4. Weakness and wasting of the *trunk*, *shoulder* and *pelvic girdle* muscles results in:
   a) **Winging of the scapulae** due to weakness of the serratus anterior.
   b) **Pot-belly abdomen** due to weakness of the abdominal muscles.
   c) **Exaggerated lumbar** lordosis due to weakness of the extensor muscles of the trunk. (the patient will extend his back to prevent himself from falling forwards by the effect of gravity).
   d) **Waddling** gait due to weakness of the gluteus medius & minimus (abductors of the hip).
   e) Climbing test or Gower's sign: *due to weakness of the gluteus maximus:*
      - The patient climbs himself in getting up from the floor.
   f) Inability to climb the stairs.
5. There is **selectivity** of the involved muscles, **e.g.** there is atrophy of the sternal head of the pectoralis major with preservation of its clavicular head.
6. No sensory changes, No fasciculations, No sphincteric disturbances.
7. Associated manifestations:
   - **Skeletal deformities,** e.g. pes cavus may be present.
   - **ECG changes, and cardiomyopathy** may be present, specially in Duchenne type.

CLINICAL TYPES *of Progressive Muscular Dystrophy*

1. Shoulder girdle types:

a) Scapulo-humeral type (Erb).

b) Facio-scapulo-humeral type (Landouzy and Dejerine).

2. Pelvic girdle types:

a) Pseudo-hypertrophic type:

- Duchenne: Severe & starts in the 1st decade of life.
- Becker: Benign & starts in the 2nd decade of life.

b) Atrophic type (Leyden-Mobius).

3. Other rare types:

a) Distal type of Gower. b) Oculo-pharyngeal type.

	Duchenne	Becker
1. Age of onset	1st decade	2nd & 3rd decades
2. Course	Progressive	Slowly progressive
3. Skeletal deformities	Present	Absent
4. ECG changes	Commonly present	Absent
5. Inheritance	X-linked	X-linked

INVESTIGATIONS

1. **Estimation of creatine and creatinine in urine:**
   - Creatine (normally absent in urine): Will appear in urine.
   - Creatinine (normally present in urine): Will decrease in urine.
2. **Estimation of serum enzymes:**
   - Elevated CPK, Aldolase & SGOT, due to DEGENERATION of the muscles.

3. **Creatine tolerance test:**
   - Administration of 2 g creatine by mouth is followed by excretion of excess amount of creatine in urine denoting lack of the ability to retain creatine and lack of ability to convert creatine to creatinine in cases of myopathy.

4. **EMG:**
   - Diminished amplitude and duration of motor units.

5. **Muscle biopsy:**
   - Degeneration of muscle fibres and its replacement by fibro-fatty tissue.

TREATMENT

There is no specific treatment, only supportive:

1. Vitamins: especially Vitamin E.
2. Physioterapy.

Causes of death in myopathy

1) Paralysis of respiratory muscles.
2) Infections specially hypostatic pneumonia.
3) Cardiomyopathy in Duchenne type.

BRAIN TUMOURS

CLASSIFICATION

I. Primary Tumours:

- From the meninges: Meningiomas.
- From the brain tissue: Gliomas.
- From the pituitary: Pituitary tumours.
- From the cranial nerves: Acoustic neuroma (of the 8th nerve).
- From the blood vessels: Hemangiomas & Hemangioblastomas.

II. Secondary Tumours:

- Metastasis.

CLINICAL PICTURE

1. **Manifestations of Increased ICT.**
2. **True localising manifestations.**
3. **False localizing manifestations.**

1. Manifestations of Increased ICT

1. HEADACHE

Due to stretch of the meninges

- Dull aching.
- Severe in the **morning** & then tends to diminish.
- Increases by: *coughing, sneezing & straining.*
- It has no significant value in localization of the site of the tumour.

2. VOMITING

Due to stimulation of the vomiting centre

- Projectile: not related to meals & not preceeded by nausea.
- More frequent in the **morning**.

3. PAPILLOEDEMA

A reliable sign of increased ICT

- Blurring of vision occurs followed by gradual diminution of vision.
- Failure of vision occurs due to post-papilloedemic optic atrophy.
- Ophthalmoscopically there will be:
  - Blurring of the margins of the optic disc.
  - Retinal hemorrhages & exudates.
  - Optic atrophy.

2. True Localizing Manifestations

- These are ***specific*** symptoms & signs that depend on the ***specific*** site of the tumour.

1. **FRONTAL LOBE TUMOURS.**
2. **PARIETAL LOBE TUMOURS.**
3. **TEMPORAL LOBE TUMOURS.**
4. **OCCIPITAL LOBE TUMOURS.**

} Refer to: areas of the Cerebral cortex

5. PITUITARY TUMOURS:

A. Suprasellar Tumours: Craniopharyngioma

1. Hypothalamic syndrome: Diabeted insipidus, obesity & hypersomnia.
2. Panhypopituitarism.
3. Bitemporal hemianopia.

B. Intrasellar Tumours:

1. Hormonal manifestations:

- Chromophobe adenoma: Panhypopituitarism.
- Acidophil adenoma: Gigantism or Acromegaly.
- Basophil adenoma: Cushing's syndrome.

2. Headache: *it passes through 3 stages*

- It starts bitemporal due to increased intrasellar pressure.
- It then disappears due to rupture of the sella turcica.
- It reappears later to produce generalized headache of ↑ ICT.

3. Neurological manifestations: *due to compression of adjacent structures*

I. Anterior comression:
- Comression of the optic chiasma: Bitemporal hemianopia.
- Comression of the optic nerve: Optic atrophy.
- Compression of the olfactory tract: Anosmia.

II. Posterior compression:
- Compression of the upper brain stem: Bilateral pyramidal signs. Ophthalmoplegia.

III. Lateral compression:
- Comression of the optic tract: Homonymous hemianopia.
- Compression of cavernous sinus: Paralysis of 3rd , 4th & 6th cranial nerves. Affection of ophthalmic division of 5th cranial nerve.

IV. Superior compression:
- Compression of the hypothalamus: Hypothalamic syndrome.

6. CEREBELLO-PONTINE ANGLE TUMOURS:

- Ipsilateral ataxia.
- Ipsilateral affection of: 5^{th}, 7^{th}, 8^{th} cranial nerves.
- Contralateral hemiplegia (pyramidal compression in the pons).

3. False Localizing Manifestations

- These are ***nonspecific*** symptoms & signs that occur regardless of the ***specific*** site of the tumour.

1. Dilatation of the lateral ventricle:

- Mental confusion.

2. Dilatation of the 3^{rd} ventricle:

- Hypothalamic syndrome (compression of the hypothalamus).
- Panhypopituitarism (compression of the pituitary gland).
- Bitemporal hemianopia (compression of the optic chiasma).

3. Dilatation of the 4^{th} ventricle: *" irritation of centers "*

- Disturbance in HR & BP (irritation of the vasomotor centre).
- Disturbance in respiration (irritation of the respiratory centre).
- Vomiting (irritation of the vomiting centre).

4. Cranial nerve paralysis:

- The most commonly affected nerve is the 6^{th} nerve as it is thin & runs a long course.

5. Herniation syndromes:

a) Herniation of the uncus of the Temporal lobe:

- Compression of the reticular formation in the MB: impaired consciousness.
- Compression of the 3^{rd} nerve nucleus in the MB: dilated fixed pupil.

b) Herniation of the cerebellar tonsils:

- Compression of the medulla: disturbance in respiration.

INVESTIGATIONS

1. MRI & CT scan:

- The best & most accurate investigations.

2. Plain X-ray of the skull:

a) Signs of increased ICT: **3S**

- **S**ilver-beaten appearance: *finger prints appearance.*
- **S**eparation of the cranial sutures.
- **S**ellar changes (in pituitary tumours): *enlargement of the sella turcica.*

b) Signs denoting the site of the tumour:

- Localised calcification.
- Localised erosion.

3. Cerebral angiography:

- Abnormal vessels feeding the tumour.

4. Air or myodil ventriculography:

- Filling defect.
- Displacement of the ventricular system.

5. Ophthalmoscopic examination:

- Papilloedema.

TREATMENT

- Surgical removal of the tumour, if possible.

EPILEPSY

DEFINITION

- It comes from the GREEK name "**Epilepsia**" which means " ***taking hold of*** or ***seizing*** ".
- It is a disorder characterized by: spontaneous tendency for recurrent ***seizures.***

SEIZURES

Recurrent transient attacks of:	***somatic***, ***psychic***, or, ***autonomic*** clinical features.
Represent:	clinical features of abnormally hyperexcitable cortical neurons.
Result from:	***paroxysmal*** and ***excessive*** electrical neuronal discharges.
ASSOCIATED WITH:	EEG changes & may be disturbance of consciousness.

ETIOLOGY

same causes of convulsions

1. Idiopathic epilepsy

no cause can be detected

- It is the commonest cause. (65 %)
- It may be associated with positive family history in some cases.
- It starts in the 1st & 2nd decades in the form of:
  - Grand mal epilepsy.
  - Petit mal epilepsy.
  - Myoclonic epilepsy.
  - Atonic seizures.

2. Secondary epilepsy

a cause can be detected

A. Local causes in the brain:

1. Congenital: cerebral palsy.
2. Traumatic: cerebral contusion or laceration.
3. Inflammatory: encephalitis, meningitis, brain abscess.
4. Neoplastic: brain tumours.
5. Degenerative: presenile dementia.
6. Vascular: stroke (especially hemorrhagic), hypertensive encephalopathy.

B. General causes with secondary effects on the brain:

1. Toxic:	- Alcohol, cocaine, lead.	- Botulism, tetanus.	
2. Iatrogenic:	- Lidocaine, INH.	- Ambilhar, Amphetamine, Aminophylline.	
3. *Metabolic*:	- ↑ glucose & ↓ glucose.	- ↑ Ca & ↓ Ca.	- ↑ Na & ↓ Na.
4. *Endocrinal*:	- Hypoparathyroidism.	- Hyperthyroid crisis.	
5. Organ failure:	- Hepatic failure.	- Renal failure.	
6. Heart disease:	- Adam's Stoke's attacks.	- Fallot's tetralogy.	
7. *Nutritional*:	- Pellagra.	- Vitamin B6 deficiency.	
8. *Physical*:	- High fevers.	- Heat stroke.	

9. HYSTERICAL.

CLINICAL PICTURE

1. GENERALISED SEIZURES

" Excessive electrical discharges from cortical neurons in BOTH hemispheres simultaneously "

I. Grand Mal Epilepsy: *" attacks of tonic-clonic convulsions "*

1. Pre-ictal stage **(aura)**
   - It is a warning sign of a coming attack.
   - It may be:
     - Somatic: *Myoclonus, Parasthesias.*
     - Psychic: *Hallucinations.*
     - Autonomic: *Tachycardia, Sweating.*
2. Ictal stage **(seizure)**
   - Sudden loss of consciousness: for seconds to minutes.
   - Tonic phase *(few seconds)*
     - The UL & LL: are extended.
     - The HEAD: is retracted to one side & the eye balls rolled up.
     - The JAWS: are firmly clenched, with biting of the TONGUE.
     - CYANOSIS: due to impaired respiration.
     - There may be incontinence of urine.
   - Clonic phase *(few minutes)*
     - The UL & LL: contract & relax repeatedly & rapidly.
     - The HEAD: jerks forcibly.
3. Post-ictal stage **(sequelae)**
   - It may be:
     - Somatic: Todd's paralysis (< 24 hours, due to neuronal exhaustion).
     - Psychic: Confusion.
     - Autonomic: Vomiting.

Drug of choice: Carbamazepine (Tegretol) or Phenytoin (Epanutin)

II. Petit Mal Epilepsy: *" attacks of loss of consciousness "* " Absence "

1. It **starts** in childhood & **improves** at puberty & usually **disappears** at the age of 20.
2. It is NOT PRECEEDED by aura & NOT FOLLOWED by sequelae.
3. It is usually PRECIPITATED by: *hyperventilation* or *photic stimulation.*
4. It is characterized by: sudden loss of consciousness of short duration (few seconds).
5. It may be associated with:
   - High frequency (50 attacks / day).
   - Falling to the ground without warning.
   - Jerky movements of the head & UL (myoclonic petit mal).

Drug of choice: Valproate (Depakine) or Succinimide (Zarontin)

III. Myoclonic Seizures: *" attacks of involuntary clonic movements "*

- It is characterized by: sudden, jerky, shock-like INVOLUNTARY muscle contraction.
  - The jerks are bilateral contractions, mainly of the shoulders and arms.
  - However, some patients report jerking in the lower limbs, trunk, or head.
- It may be of 2 types:
  - Simple: - Occurs singly (no loss of consciousness).
  - As a part of: - Grand mal epilepsy (aura). - Petit mal epilepsy.

Drug of choice:	**Valproate (Depakine)**	**or**	**Clonazepam (Rivotril)**

IV. Atonic seizures:

- Transient attacks of brief loss of postural tone, often resulting in falls and injuries.

2. PARTIAL SEIZURES

" Excessive electrical discharges from cortical neurons in a certain area in ONE hemisphere "

A. Simple seizures: " No disturbance in consciousness "

- The CP depends on the site of the hyperexcitable neurones in the cerebral cortex, whether in: *"Motor area or Sensory areas"*.

1. Motor fits:
   - Focal fits: movement of part of a limb or the whole limb.
   - Motor jacksonian fits: movement of one side of the body (see before).
2. General Sensory fits:
   - Focal fits: parasthesia of part of a limb or the whole limb.
   - Sensory jacksonian fits: parasthesia of one side of the body (see before).
3. Special Sensory fits:
   - Visual hallucinations: irritation of the visual sensory area.
   - Auditory hallucinations: irritation of the auditory sensory area.
   - Olfactory hallucinations: irritation of the uncus.

B. Complex seizures: " disturbance in consciousness "

- SITE: The hyperexcitable neurons are in the Temporal lobe "Temporal lobe epilepsy".
- DURATION: The seizure lasts few seconds to few minutes.
- The seizure starts with **A**ura, followed by **A**bsence, **A**utomatism, **A**mnesia:

1. **A**ura: Olfactory hallucinations, Déjà-vu phenomenon, Sensation of fear.
2. **A**bsence: Absent patient with staring eyes (with no response to conversation).
3. **A**utomatism: Involuntary Purposeless acts: ***motor*** (eg, lip smacking, chewing) or ***verbal***.
4. **A**mnesia: No recalling of the seizure.

3. PARTIAL SEIZURES → GENERALISED SEIZURES

" Partial seizures may spread to involve the whole brain → secondarily generalised seizures " .

Hysterical hemiplegia

- Usually: young neurotic ♀.
- The cause: psychological & there is no organic lesion.
- Incidence: usually occurs in the presence of people.
- It is associated with: *anxiety, palpitaion & hyperventilation.*
- It is not associated with: *tongue biting* or *incontinence of urine.*
- EEG: normal.

PRECIPITATING FACTORS

- **M**issed ttt.
- **M**enses.
- **A**lkalosis.
- **A**lcohol use & Drug abuse (e.g. cocaine).
- **S**timulation by photons & Hyperventilation.
- **S**leep deprivation & Stress & sudden withdrawal of antiepileptic drugs.

INVESTIGATIONS

1. EEG:

- It is the most specific test for epilepsy because it records the electrical activity of the brain.
- It shows specific pattern: " *Epilepsy waves* ".

2. LOCAL INVESTIGATIONS: "CT & MRI of the brain"

- To identify or exclude a LOCAL CAUSE of seizures in the brain.

3. GENERAL INVESTIGATIONS: "Laboratory investigations"

- To search for a GENERAL CAUSE of seizures, e.g. *blood glucose*.

TREATMENT

A. General Measures:

1. Moderation of the patient's physical activity.
2. Avoid the precipitating factors (Alcohol, hyperventilation, photic stimulation.....).
3. A ketogenic diet is encouraged because it will induce acidosis:
   - ***Acidosis is beneficial as it raises the threshold of stimulation of the brain cells.***

B. Specific Treatment:

1. Treatment of the cause in secondary epilepsy.
2. Anti-epileptic drugs: *" General rules for use ":*
   a) Always start with one drug, then add another drug if there is no response.
   b) Always stop the drugs ONLY if:
      - The patient stays free of symptoms for at least 2 years.
      - The patient has a normal EEG.
3. Side effects of Anti-epileptic drugs:
   1. Skin rash.
   2. Bone marrow depression.
   3. Ataxia.

ANTI-EPILEPTIC DRUGS

Drug	Dose	Best indicated
1. Barbiturates (Phenonobarbitone)	100-600 mg / day	- Broad spectrum. - Not for petit mal.
2. Hydantoin (Epanutin)	100-600 mg / day	- Grand mal. - Motor Jacksonian fits.
3. Carbamazepine	200-600 mg / day	- Grand mal. - Motor Jacksonian fits. - Complex seizures. - Not for petit mal.
4. Clonazepam	2 – 6 mg / day	- Myoclonic. - Grand mal.
5. Valproate	500-1500 mg / day	- Broad spectrum.
6. Succinamide	500-1000 mg / day	- Petit mal.

NEW ANTI-EPILEPTIC DRUGS

- These drugs are new drugs that may be used in ***resistant seizures***.

1. Lamotrigine: 200 – 400 mg / day.
2. Felbamate: 400 – 800 mg / day.
3. Gabapentin: 600 – 1200 mg / day.

STATUS EPILEPTICUS

DEFINITION

- A medical emergency where there are:

1. *Repeated attacks of generalized convulsions, with lack of recovery of consciousness,* OR,
2. *Persistent attack of seizure lasting for at least 30 minutes.*

- If the convulsions are not stopped rapidly, coma deepens & death may occur due to:
  heart failure or **respiratory failure** or **brain damage**.
- The most common causes are: sudden withdrawal of anti-epileptic drugs & stroke.

TREATMENT

A. General Measures:

1. Take care of: **" ABC "**
   - Place the patient on the ground, to guard against falling from bed.
   - Mouth gag & O_2 inhalation (endo-tracheal intubation may be needed).
   - Record the vital signs regularly.

2. Take a sample of:
   - Venous blood: for the level of: **a**nti-epileptic drugs, **a**lcohol.
   - Arterial blood: for the level of: pH, pO_2, pCO_2, HCO_3.

3. Give cerebral dehydrating measures: e.g. *Lasix, conc. Mannitol, Dexamethazone.*

B. Specific Treatment:

- ***Epanutin with Valium (or Rivotril) are given immediately:***

1. EPANUTIN (Phenytoin): 15 mg / Kg slow infusion.
2. VALIUM (Diazepam): 5 mg slowly IV, to be repeated after 5 minutes if seizures recur: maximum dose: 20 mg.

OR:

RIVOTRIL (Clonazepam): 2 mg slowly IV, to be repeated after 5 minutes if seizures recur: maximum dose: 6 mg.

- ***If seizures persist after 20 min. of Epanutin & Valium:***

3. PHENOBARBITONE: 200 mg infusion.

- ***In resistant cases:***

4. GENERAL ANAESTHESIA: may be used.

DISSEMINATED SCLEROSIS (DS)
MULTIPLE SCLEROSIS (MS)

DEFINITION

- It is an INFLAMMATORY disease of the CNS (Brain & Spinal cord).
- It affects mainly the WHITE MATTER in the form of patchy DEMYELINATION.

ETIOLOGY

1. AUTO-IMMUNE: *CSF shows increase in immunoglobulins especially IgG.*
   - The immune system attacks its own **CNS**, leading to **DEMYELINATION**.

   - ANTIBODIES occur against proteins in the myelin sheath surrounding the nerves.
   - This causes inflammation and injury to the sheath and ultimately to the nerves.
   - The result may be multiple areas of **scarring** (**SCLEROSIS**).
   - The damage slows down the nerve signals leading to impairment of the function.

2. Less accepted old theories: Viral infection or Ischemia of the white matter.

RISK FACTORS

1. **I**NHERITANCE: The disease runs in families.
2. **I**NFECTIONS: Viral or Bacterial **infections** may trigger the disease.
3. *Pregnancy & Labour.*
4. **S**TRESS: Physical & Emotional.
5. **S**URGERY & TRAUMA.

CLINICAL PICTURE

TYPE OF THE PATIENT

Age: 20-40 years. **Sex:** more common in FEMALES .

BEHAVIOUR OF THE DISEASE

Onset: usually acute. **Course:** REMISSIONS & EXACERBATIONS.

FEATURES: *any of the following manifestations:*

1. **Mentality changes:**
   - Euphoria or Depression or Emotional lability - Cognitive dysfunction (memory loss).
2. **Speech disturbance** (Dysarthria):
   - Staccato speech - Slurred speech.
3. **Cranial nerve affection:** **2, 3, 7, 8**
   - Optic: **2** Optic neuritis, ending in BLINDNESS.
   - Oculomotor: **3** Ophthalmoplegia with diplopia & squint.
   - Facial: **7** UMNL (commonly), LMNL (rarely).
   - Cochleo-vestibular: **8** Vertigo.
4. **Motor system affection** (Pyramidal affection):
   - **Monoparesis, Paraparesis**, hemiparesis, quadriparesis, - Pseudo-bulbar palsy.
5. **Sensory system affection** (Posterior column affection mainly):
   - Initially: parasthesias, Late: sensory loss (S or D) & **sensory ataxia.**
   - L'hermite sign: Electric-like sensation felt in the back & limbs on bending the neck. It is due to posterior column affection in the cervical region.
6. **CEREBELLAR AFFECTION:** (COMMON MANIFESTATION)
   - Features of cerebellar ataxia.
7. **Autonomic affection:**
   - Sphincteric troubles: *precipitancy or retention.* - Impotence.

CLINICAL TYPES

1. **Benign:** **10 %**
   Symptoms are mild to moderate, do not worsen and do not lead to permanent disability.
2. **Relapsing remitting:** **75 %**
   One or two flare-ups occur every few years, followed by periods of remission.
   Flare-ups typically **appear** acutely, **last** a few weeks or months, then gradually **disappear**.
3. **Progressive:** **15 %**
   - ***Primary progressive:***
     After symptoms first appear, continuous deterioration occurs without periods of remission.
   - ***Secondary progressive:***
     After years of relapsing remitting MS, continuous deterioration occurs.

EARLY MANIFESTATIONS

1. EYE: Optic neuritis.
2. AUTONOMIC: Sphincteric troubles & Impotence.

INVESTIGATIONS

1. Fundus examination:

- Pallor of the optic disc.

2. CSF examination:

- Cells: Marked increase up to 50 cells / mm^2 *mainly lymphocytes.*
- Proteins: Moderate increase.

The most sensitive & specific findings in CSF

- Increase in **IMMUNOGLOBULIN** concentration, *especially IgG.*
- Presence of **OLIGOCLONAL BANDS** on *protein electrophoresis.*

3. Cortical Evoked Responses: "CER"

Normally: Stimulation of any sensory receptor (visual, auditory, or somatosensory) evokes an electrical signal in the corresponding region of the cerebral cortex "CER".

In MS: Stimulation of any sensory receptor (visual, auditory, or somatosensory) evokes a **slow** or **abnormal** CER due to loss of myelin (↓ nerve impulse conduction).

Therefore: Recording of CER may help in detection of demyelinated lesions in MS.
e.g. Abnormal Visual Evoked Potential (VEP) = lesion in the visual pathway.

4. IMAGING:

I. MRI:

Most important investigation

A. Confirms the diagnosis:

Patchy multiple areas in the white matter (areas of demyelination).

B. Differentiates new lesions from old lesions: MRI with Gadolinium

In new lesions of MS, recent inflammation leads to increased vascular permeability; this is detected by leakage of IV contrast agent Gadolinium into the brain on MRI.

II. CT scan:

Patchy multiple areas in the white matter (areas of demyelination).

TREATMENT

No curative ttt

1. Immunomodulatory drugs: " A B C "

A **A**vonex: interferon ßeta -1a.

B **B**etaseron: interferon ßeta -1b.

C **C**opaxone.

- *They reduce:* *the frequency & severity of the attacks.*
- *They delay:* *the progression to disability.*

2. Immunosuppressive drugs: " M A C "

a) **M**ethotrexate. b) **A**zathioprine. c) **C**yclophosphamide.

3. TTT of acute exacerbations:

I. Corticosteroids:

Give:

a) Methyl prednisolone: 1 gm IV infusion / day for 1 week ***OR***:

b) ACTH: 80 IU given IM / day for 1 week, ***then:***
40 IU given IM / day for another week.

Continue with:

- Prednisone: 1 mg / Kg / day orally for 1 week, then: taper gradually.

II. Plasmapheresis: *may be of value in cases not responding to corticosteroids.*

4. Symptomatic TTT, Physiotherapy & Vitamins.

HEADACHE

DEFINITION

Mild to severe pain in the head including the FRONT of the face & the BACK of the head.

ETIOLOGY & TYPES

1. Vascular headache: *due to Vascular dilatation or Vasculitis*

a) Primary: MIGRAINE, Cluster headache, Temporal arteritis.

b) Secondary:

- Hypertension.
- Hypoxia.
- Hypoglycemia.
- Drugs causing VD: e.g. Nitrates.
- Toxins causing VD: Alcohol, Infections (bacterial or viral toxins).

2. Inflammation:

- Intracranial: Meningitis, Encephalitis.
- Neuritis: *of the sensory nerves of the scalp: e.g.* Trigeminal neuralgia.

3. Traction headache: *due to stretch of the meninges, ↑ICT*

- Brain tumour, abscess, or hemorrhage.
- Post-lumbar puncture.

4. Muscle contraction headache:

When one gets a " STIFF NECK," the prolonged muscle spasm can compress the nerves and also induce ischemia with production of metabolites which stimulate pain receptors:

- Prolonged driving.
- Cervical spondylosis.

5. Referred headache: *due to diseases in different parts of the head*

- Eyes: e.g. glaucoma.
- Ears: e.g. ear infections
- Nose: e.g. sinusitis
- Mouth: e.g. dental sepsis.

6. Tension headache (Psychogenic): *"the most common cause"*

- It occurs in: psychologically disturbed persons usually FEMALES.
- It occurs as: pressure or BAND-LIKE, Bilateral fronto-occipital.

CRITERIA OF A SERIOUS HEADACHE

1. SUDDEN, severe headache.
2. Headache asscociated with projectile vomiting & blurring of vision: ↑ ICT.
3. Headache that begins after an INJURY to the head.
4. Headache followed by DCL or associated body weakness.
5. Headache associated with FEVER and a stiff neck.

MIGRAINE

DEFINITION

- It is a paroxysmal disorder characterized by intense throbbing headache, (usually unilateral) associated with autonomic manifestations, e.g. nausea & vomiting.

ETIOLOGY

- Idiopathic.
- GENETIC: Heredo-familial (80 %).

INCIDENCE

- SEX: more in young FEMALES (female / male = 3 / 1).
- RESIDENCE: more in urban areas.
- PERSONALITY: more in perfectionistic people.

MIGRAINE TRIGGERS "4 M"

- **M**ental & physical exertion.
- **M**enses.
- **M**edications: *Vasodilators (nitrates), CCPs.*
- **M**eals (& .....): *Cheese, Chocolate, Citrus fruits. Smoking, Alcohol.*

CLINICAL PICTURE

2 stages

1. First stage: " Aura " due to VC

- Visual: Flashes of light or blind spots.
- Sensory: Parasthesias.
- Motor: Weakness.

2. Second stage: " Migraine " due to VD

It occurs in periodic & recurrent attacks:

- SITE & REFERENCE: **Starts** in the temple or around the eye & then: **Spreads** to involve the whole side of the head.
- CHARACTER: Pulsating or Throbbing.
- DURATION: It lasts for few hours to few days (3hours to 3 days).
- AGGRAVATED BY: Bright light, Physical & Emotional stress.
- RELIEVED BY: Sleep.
- ASSOCIATIONS: Nausea, vomiting, Photophobia, Phonophobia.

PATHOGENESIS

1. NEUROVASCULAR THEORY

A) VASOACTIVE NEUROPEPTIDES

- Migraine triggers → abnormal overexcitation of Trigeminal nerve axons → release of VASOACTIVE NEUROPEPTIDES including Substance P from Trigeminal nerve endings (near the meningeal blood vessels).

B) VASODILATATION + STERILE INFLAMMATION

- These peptides will cause: VD + STERILE INFLAMMATION at the Trigeminal nerve endings that spreads to the nearby meninges.

C) PAIN results due to both VD & INFLAMMATION.

Serotonin through acting on its receptors appears to block these peptides

2. SEROTONIN THEORY:

Initially: An initial increase in plasma serotonin levels will cause constriction of cerebral blood vessels and a reduction in cerebral blood flow → AURA.

Later: A subsequent drop in plasma serotonin levels will then lead to marked dilation of the arteries → HEADACHE of MIGRAINE.

3. VASCULAR THEORY "OLD"

Initially: An unexplained VC (hypoperfusion) begins in the visual area and spreads in the cerebral cortex for a short time → AURA.

Later: Reactive VD follows → HEADACHE of MIGRAINE.

4. DOPAMINE THEORY

Recently: abnormal Dopaminergic stimulation → HEADACHE of MIGRAINE, and also causes associated manifestations such as nausea & vomiting.

TYPES

1. CLASSIC migraine (15 %): always preceded by aura.
2. COMMON migraine (85 %): not preceded by aura; probably due to mild VC stage.
3. RARE VARIANTS:
   - *Hemiplegic migraine:* *associated with transient hemiplegia.*
   - *Facial migraine:* *associated with transient facial paralysis.*
   - *Ophthalmoplegic migraine:* *associated with transient ophthalmoplegia.*

TREATMENT

A. During the attack:

1. SEROTONIN AGONISTS:

They act on the serotonin receptors in the cerebral blood vessels (which were already dilated) leading to their VC & relief of headache.

a) Selective agonists: SUMATRIPTAN

- Orally: Single tab (50 or 100 mg), may be repeated after 2 hours, Maximum daily dose: 200 mg.
- Parentrally: 6 mg, single SC injection, may be repeated after 2 hours, Maximum daily dose: 12 mg (2injections).
- Nasal spray.

b) Non-Selective agonists: ERGOTAMINE

- Orally: Ergotamine, 1 mg, 2 tablets once.

  Ergotamine 1 mg + Caffeine 100 mg (added caffeine will enhance absorption of ergotamine & potentiate its VC effect).

2. DOPAMINE ANTAGONISTS: METOCLOPRAMIDE (orally, IV)

- Adjunctive therapy in an acute attack of migraine.
- Relieves associated nausea & vomiting (antiemetic effect).

3. ANALGESICS: NSAIDs.

B. Inbetween the attacks: "Prophylactic"

1. **Avoid Migraine triggers.**

2. **SEROTONIN ANTAGONISTS:**
   - Pizotifen: 0.5 mg tds.
   - Methysergide: 1 mg tds.

3. **CALCIUM CHANNEL BLOCKERS:**
   - Sebelium: 10 mg / day before sleep.
   - Verapamil: 80 mg tds.

4. **ßETA-BLOCKERS:**
   - Propranolol:

     Action: It prevents the uptake of adrenaline by the receptors in the vessel wall.

     Dose: 10 mg t.d.s.

Beta-blockers are the ttt of choice in patients whose migraine attacks are related to stress

5. **OTHER MEASURES:**
   - Antihistaminics.
   - Antidepressants.
   - Antiepileptics.

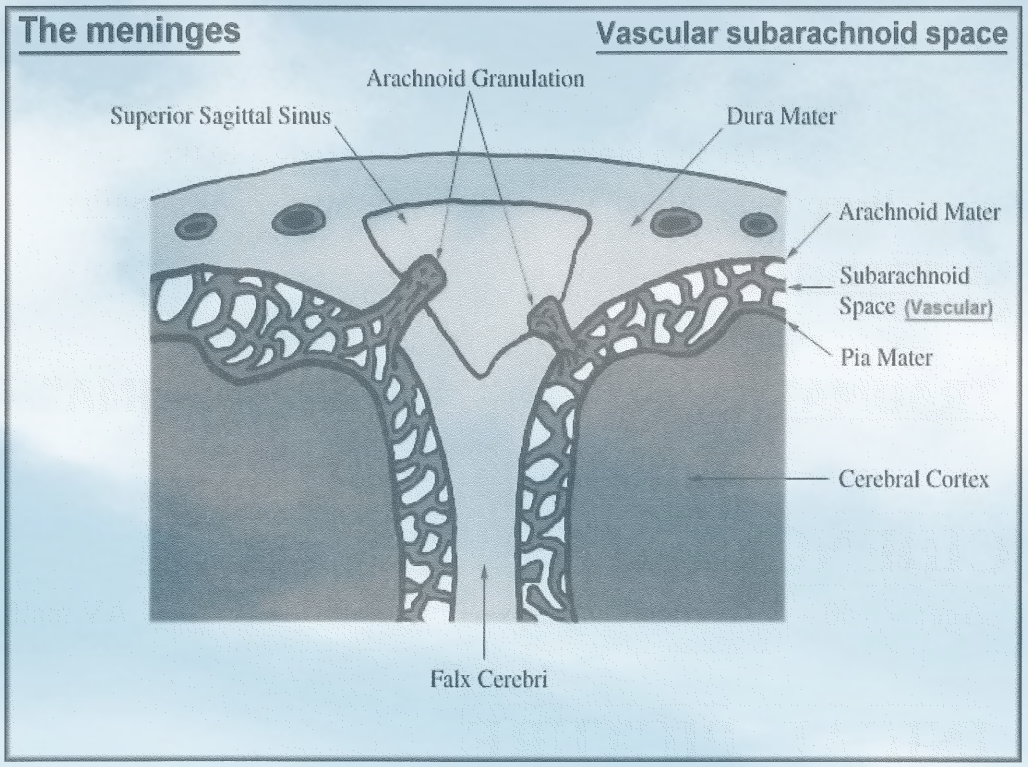
Cluster Headache

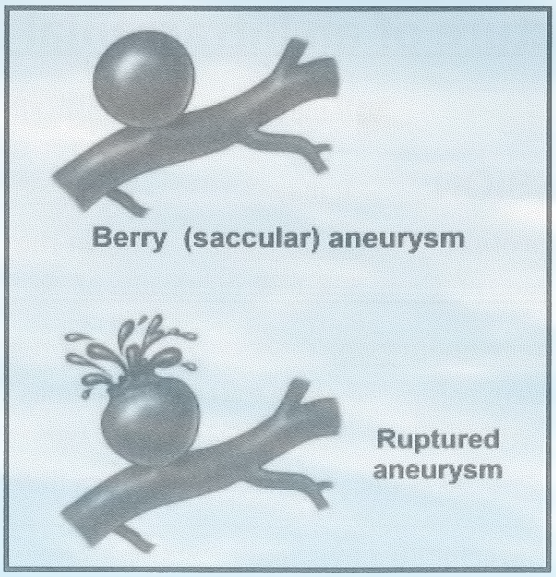
- **Character:** attacks of severe headache starting on one side in the orbital & frontal region & spreading gradually to the same side of the head & neck.
- **Duration:** several minutes to one hour.
- **Frequency:** the attacks occur in clusters, i.e. every 24 hours for weeks or months. the clusters are followed by long free periods (6 months or one year).

SUBARACHNOID HAEMORRHAGE

DEFINITION

- SAH means bleeding into the subarachnoid space which the space between:
  The ***arachnoid membrane*** and the ***pia mater***.





ETIOLOGY

I. SPONTANEOUS

1. RUPTURE

- Intracranial aneurysm **[Berry]**: *"most common cause"* "80 %"
  a. Congenital. *b. Atherosclerotic.* *c. Mycotic* (e.g. in SBE).
- Intracranial AV malformation.

2. HEMORRHAGE

- Intracerebral Hemorrhage: that extends to the subarachnoid space.
- Hemorrhage in a brain tumour: that extends to the subarachnoid space.
- Hemorrhagic blood diseases: *purpura*, *hemophilia*.

3. HYPERTENSION.

II. TRAUMATIC "HEAD TRAUMA"

INCIDENCE

- Age: 40 – 60 in case of aneurysm, 10 – 20 in case of AV malformation.

CLINICAL PICTURE

I. Clinical picture of an intracranial aneurysm:

1. SILENT:

It may: remain **silent** with no manifestations.

2. COMPRESSION:

It may: **compress** neighbouring structures, **e.g.**

a. Internal carotid artery aneurysm: in the cavernous sinus:
- Compression of Cr. n. 3, 4, 6 & ophth. div. of 5: *ophthalmoplegia & facial pain.*

b. Basilar artery aneurysm: compression of midbrain or pons:
- Cranial nerve palsies & long tract manifestations (e.g. hemiparesis).

3. RUPTURE:

90 % of cases first present when they **rupture** giving CP of SAH.

Rupture may be **precipitated** by: Strain, Stress, Severe cough or Sexual intercourse.

II. Clinical picture of subarachnoid hemorrhage:

1. Features of INCREASED ICT:

a) **Headache:** *due to irritation of the meninges by blood*

- Onset: SUDDEN.
- Character: SEVERE (**worst headache in the life**), ↑ by flexion of the neck.
- Site: STARTS at the back of the head & upper neck, THEN: generalized.
- Reference: Shoulders, back & limbs.

b) **Vomiting:** *Projectile*

c) **Papilloedema.**

2. Features of MENINGEAL IRRITATION:

a) **Pain:** *due to irritation of the spinal sensory roots*

- In the back of the neck, shoulders & upper limbs.
- In the lower back & lower limbs.

b) **Neck:** stiffness or rigidity.

c) **Body:** opisthotonus (high arched back).

d) **Positive signs of meningeal irritation.**

POSITIVE SIGNS OF MENINGEAL IRITATION

Kernig's sign: While the hip and knee joints are flexed at 90°,
Extending the knee is limited & painful due to stiffness in Hamstrings.

Lasegue's sign: While the hip and knee joints are fully extended,
Raising the leg by flexing the hip is limited & painful.

Brudzinsk's sign:

a. Neck sign: Passive flexion of the neck leads to flexion of both hips & knees.

b. Leg sign: Passive flexion of one hip leads to flexion of the other hip & knee.

3. Features of CRANIAL NERVE AFFECTION: *pressure by blood*

The most commonly affected nerves are the ocular nerves (3, 4 , 6) & the optic nerve (2).

4. Features of EYE AFFECTION:

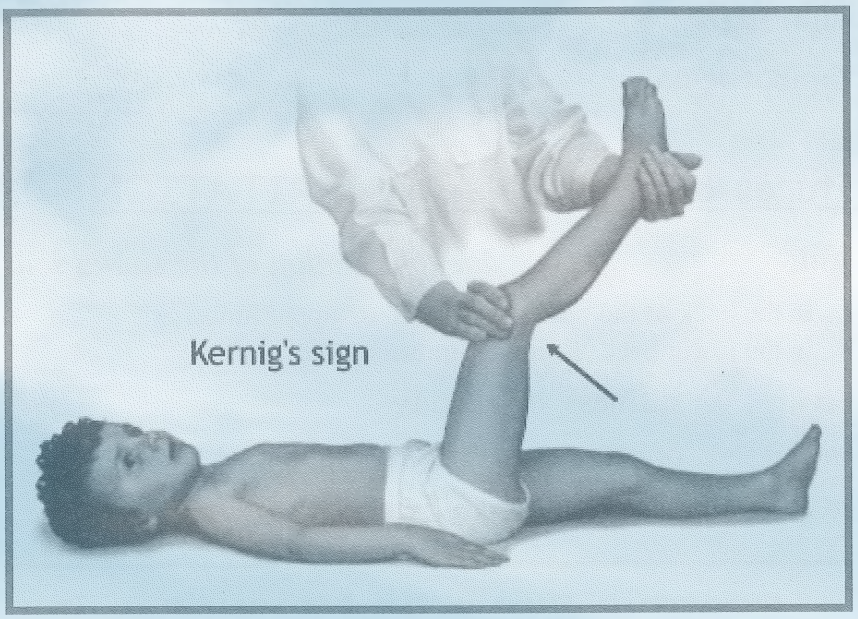
a) **Flame-like retinal hemorrhages:** due to blood in the subarachnoid sheath of optic n.

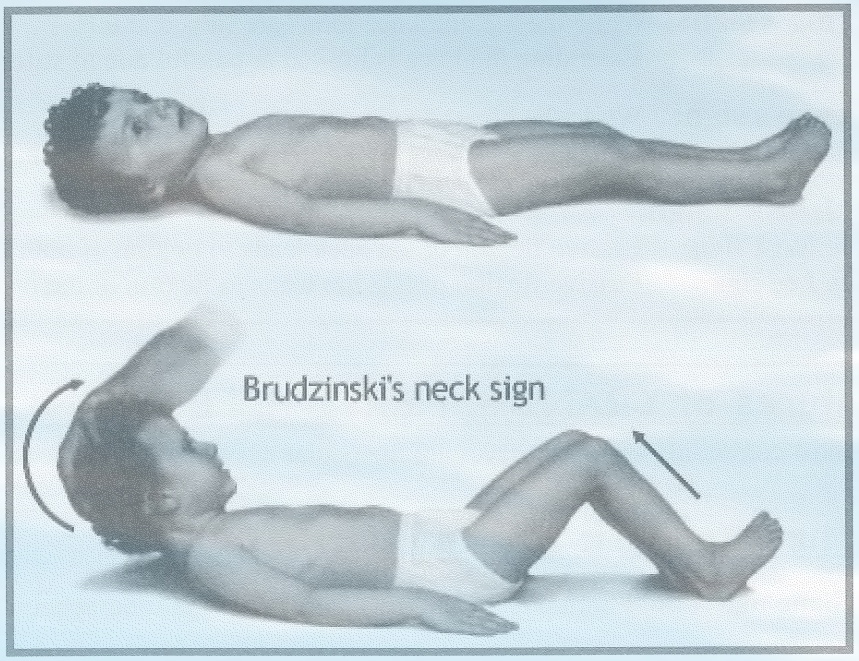
b) **Papilloedema:** due to increased ICT.

5. Features of CEREBRAL AFFECTION: *due to pressure by blood*

- Confusion & **Coma**.
- Cortical sensory loss.
- **Convulsions**.
- Contralateral hemiplegia.
- Contralateral homonymous hemianopia.
- **Aphasia**.

Remember:
CORTICAL
HEMIPLEGIA





INVESTIGATIONS

1. CT scan or MRI: *The first investigation to be done*

- If blood is detected, the diagnosis is established and lumbar puncture is not needed.

2. CSF examination: *Lumbar puncture*

a. Pressure: high.
b. Aspect: grossly bloody.
c. Cells: markedly increased: *RBCs*.
d. Proteins: markedly increased.
e. Glucose: normal.
f. Chlorides: normal.
g. Organisms: CULTURE IS NEGATIVE.

3. Cerebral Angiography:

- Localises the site of the aneurysm or the AV malformation.

4. Plain X-ray skull:

- Calcified wall of aneurysm or AV malformation.
- Erosion or fracture of bone.
- Signs of increased ICT (in case of a brain tumour).

COMPLICATIONS

1. Rebleeding: *associated with a worse prognosis.*
2. Vasospasm: *causes cerebral ischemia or infarction.*
3. Hydrocephalus: *due to occlusion of CSF flow by the blood.*
4. Epilepsy: *due to cortical irritation and damage.*
5. HYPONATREMIA: *due to SIADH.*

DIFFERENTIAL DIAGNOSIS

1. Other causes of coma.
2. Other causes of cerebral hemorrhage.
3. Meningitis: *" Refer to DD of meningitis ".*

TREATMENT

I. MEDICAL TTT:

INDICATIONS

1. Acute cases.
2. Non-surgical cases, **e.g.** *hemorrhagic blood diseases*.
3. Difficult surgical cases, **e.g.**
   - Multiple aneurysms.
   - Huge or inaccessible aneurysms.

MEASURES

1. Avoid the precipitating facors for rupture.
2. Analgesics to relieve headache.
3. Antifibrinolytic agents: **e.g.** *aminocaproic acid*
   - They prevent clot dissolution around the aneurysm and thus prevent rebleeding.
4. Dehydrating measures for the brain: **e.g.** *IV mannitol*.
5. Lumbar puncture: *to relieve severe headache; should be done carefully.*
6. Treatment of the cause: **e.g.** ttt of Hypertensive emergency.

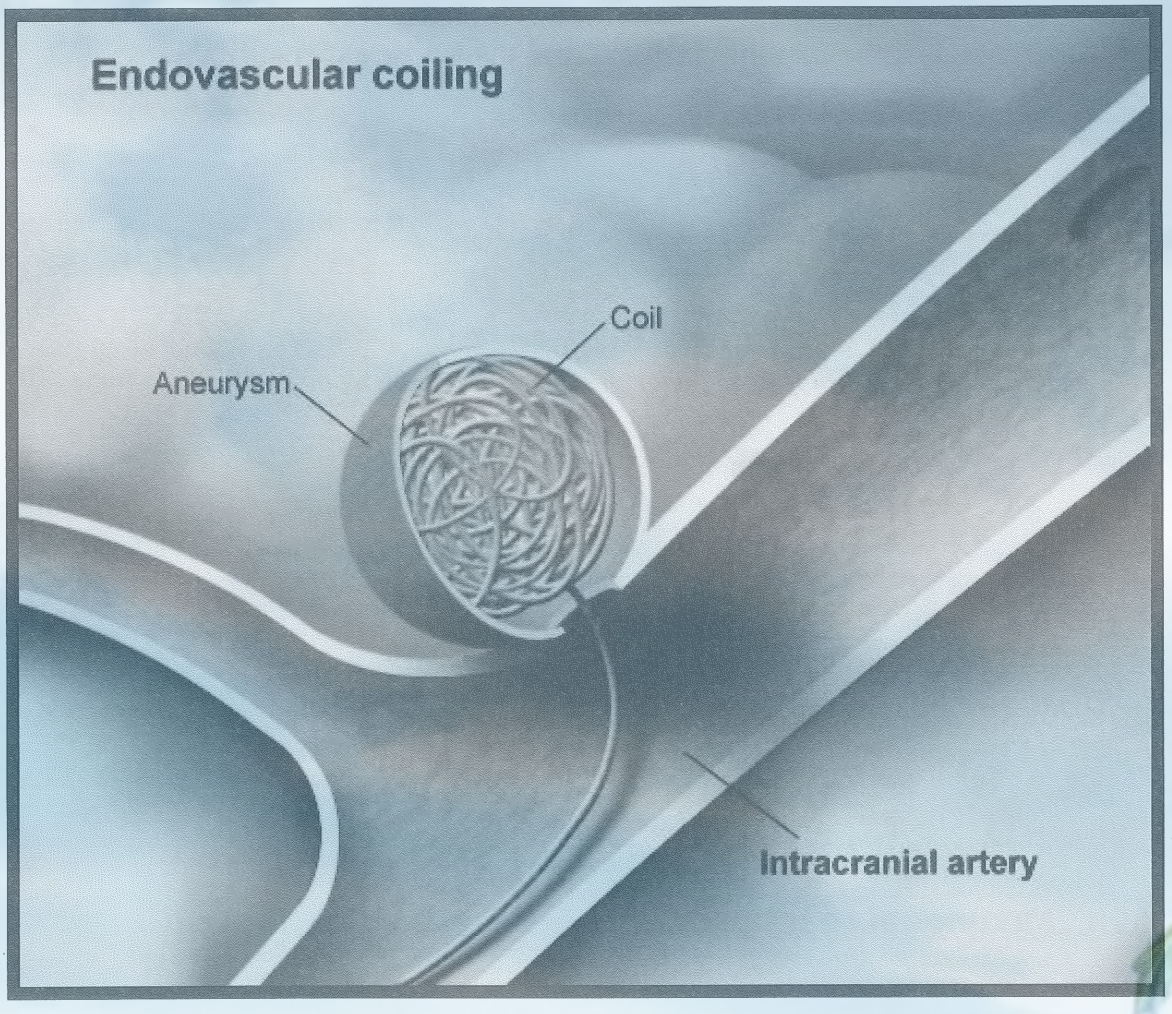
II. INVASIVE INTERVENTION:

1. Surgery:

- Direct surgery to the *aneurysm*, e.g. **Surgical clipping**.

2. Minimally invasive technique: "Endovascular coiling"

- Tiny platinum coils are threaded through a catheter and deployed into the aneurysm, blocking blood flow into the aneurysm and therefore:
  - Prevent rupture (Prophylactic)..... **or:**
  - Reduce bleeding (Therapeutic).


Endovascular coiling
Coil
Aneurysm
Intracranial artery

MENINGITIS

DEFINITION

- Inflammation of the membranes covering the CNS, especially the *pia-arachnoid.*

ETIOLOGY

1 SEPTIC MENINGITIS "Bacterial meningitis"

Acute purulent meningitis:

- The CSF contains ***mainly polymorphs*** due to infection by organisms **that form pus:**
  - Meningococci.
  - Streptococcus pneumoniae.
  - Hemophilus influenza.

2 ASEPTIC MENINGITIS "Non-Bacterial meningitis"

a. Subacute lymphocytic meningitis:

- The CSF contains ***mainly lymphocytes*** due to infection by organisms **that do not form pus:**
  - Viruses: **H**SV, **H**IV, **E**nteroviruses, **E**chovirus & Coxsackie.
  - TB: TB meningitis.
  - Fungi: Cryptococcosis, Mucormycosis.

b. Meningeal reaction:

- ***Inflammatory reaction*** of meninges **with no pus formation** due to:
  - Nearby infection: as mastoiditis.
  - Sensitivity to some medications: as ibuprofen.
  - Inflammatory diseases: as SLE.

MENINGOCOCCAL MENINGITIS

(Acute Cerebro-Spinal Fever)

ETIOLOGY

1. Causative organism: meningococci.
2. Source of infection: nasopharyngeal carriers.
3. Mode of infection: droplet infection.

PATHOGENESIS

1. **NASOPHARYNX:** After infection, the organisms localize in the **nasopharynx** causing: *catarrhal inflammation*.
2. **BLOOD:** The organisms then invade the **blood** → short meningococcemia (hours) which may be severe and cause: *the fulminating type*.
3. **MENINGES:** From the blood they reach the **meninges** → purulent exudates which collect in the subarachnoid space causing: *meningitis*.
4. The organisms may be localized in:
   - The adrenals → acute adrenal failure.
   - The skin → skin rash.

CLINICAL PICTURE

IP: 4 – 5 days

1. GENERAL signs:

- **Pyrexia:** Acute high fever (39 – 40 ° C).
- **Pulse:** Tachycardia, except in cases of ↑ ICT (bradycardia).
- **Photophobia:** Oversensitivity to bright light.
- **Papular skin rash.**

2. Features of INCREASED ICT:

a) Headache: ***due to irritation of the meninges by infection***

- Onset: ACUTE.
- Character: SEVERE, ↑ by flexion of the neck.

b) Vomiting: *Projectile*

c) Papilloedema.

3. Features of MENINGEAL IRRITATION:

a) Pain: ***due to irritation of the spinal sensory roots***

- In the back of the neck, shoulders & upper limbs.
- In the lower back & lower limbs.

b) Neck: stiffness or rigidity.

c) Body: opisthotonus (high arched back).

d) Positive signs of meningeal irritation: Kernig's sign….. etc….

4. Signs of NEUROLOGICAL DEFICITS:

- Confusion or coma.
- Convulsions.
- Transient cranial nerve paralysis : due to exudation around the nerves.
- Focal neurological signs: e.g. HEMIPLEGIA: rare.

CLINICAL TYPES

1. Acute meningococcal meningitis.
2. Fulminating type: The patient dies from marked toxemia before meningeal symptoms appear:
   i. Cerebral type: fever, coma & death.
   ii. Adrenal type: Waterhouse-Freidreichson's syndrome:
   - Massive bilateral adrenal cortical hemorrhage leading to *Addisonian crisis* & *death*.
3. Chronic meningococcal meningitis.

COMPLICATIONS

1. Neurological: Hydrocephalus due to occlusion of CSF flow by the *organized exudates*.
2. Cardiac: Pericarditis & Endocarditis.
3. Eyes: Conjunctivitis, keratitis, iridocyclitis, & may be BLINDNESS.
4. Ears: DEAFNESS due to affection of the 8th cranial nerve.
5. Others: Arthritis, nephritis, orchitis.

INVESTIGATIONS

1. CT or MRI of the brain: *to exclude subarachnoid hemorrhage.*

2. CSF examination:

a. Pressure: high.
b. Aspect: turbid, purulent.
c. Cells: markedly increased especially *POLYMORPHS*.
d. Proteins: markedly increased.
e. Glucose: markedly decreased.
f. Chlorides: moderately decreased.
g. Organisms: detected by Gram-stain or CSF culture.

3. CBC:

- Leukocytosis: with shift to the left.

4. Culture of the blood:

- The organism may be detected in many cases of blood culture.

DIFFERENTIAL DIAGNOSIS

1. Subarachnoid hemorrhage:

- ONSET: *Sudden.*
- CSF: *CSF is bloody, CSF culture is negative, CSF sugar & chloride are normal.*
- CT scan: *reveals the hemorrhage.*

2. Encephalitis:

- Presence of parenchymal involvement, e.g. *cerebrum, brain stem, cerebellum.*

3. Acute infections with cerebral symptoms: e.g. *Typhoid.*

- Absence of signs of meningeal irritaion.
- Characteristic CP of the acute infection.

4. Meningism: MENINGEAL IRRITATION

- Meningeal irritation may occur in acute infections due to increased water content of the CSF in an attempt to dilute the toxins.
- CSF: no organisms, high pressure, DILUTED: low glucose & chlorides & proteins.

5. Other causes of meningitis:

a) Other causes of acute septic meningitis: e.g. *Strept. pneumoniae, H. influenza*

- Differentiated by: CSF culture.

b) Viral meningitis:

- Course: benign.
- CSF: clear, NORMAL chloride & glucose, HIGH protein & lymphocytes.

c) TB meningitis:

- ONSET: Gradual.
- TB: Features of TB toxemia, Features of a primary TB focus.
- MENINGITIS: Minimal signs of meningeal irritation.
- CSF: Typical CSF findings: *see the table.*

Typical CSF of TB meningitis

- Pressure: high.
- Aspect: cloudy.
- Cells: increased especially *LYMPHOCYTES.*
- Proteins: markedly increased.
- Glucose: moderately decreased.
- Chlorides: markedly decreased.
- Organisms: detected by ZN stain or culture (L-J or *BACTEC*)

TREATMENT

I. Prophylactic:

1. Isolation of the patient.
2. Chemoprophylaxis: Rifampicin 600 mg bid for 2 days for contacts.
3. Immunoprophylaxis: Meningococcal Live attenuated vaccine.

II. Curative:

1. Antibacterial treatment:

- Once meningitis is suspected we should start ttt immediately even before culture:
  - **C**efotaxime (Claforan): 4 gm / day IV, OR
  - **C**rystalline penicillin: 4 million units / 6 hours IV, OR
  - **C**hloramphenicol: 100 mg / Kg / day in penicillin-sensitive patients.

2. Corticosteroids:

- In severe cases as: Waterhouse-Freidreichson's syndrome.
- In case of: TB meningitis undercover of Anti-TB drugs.

ENCEPHALITIS

DEFINITION

- Inflammation of the brain substance.

ETIOLOGY

I. Primary encephalitis: *primarily attack the CNS*

1. Neurotropic viruses: attack the nerve cells
   - Poliomyelitis. - Rabies.
2. Pantropic viruses: attack the neurones
   - Arthropode borne viruses.

II. Secondary encephalitis: *do not primarily attack the CNS*

1. Viral: Herpes simplex, Herpes zoster, Mumps.
2. Bacterial: Typhoid, Bacillary dysentery.
3. Parasites: Malaria.
4. Others: *MYCOPLASMA*, *chlamydia*, *rickettsiae*.

CLINICAL PICTURE

1. Manifestations of parenchymal involvement: *focal OR diffuse*
   - Cerebrum: coma, aphasia, convulsions, hemiparesis.
   - Brain stem: cranial nerve palsies, long tract manifestations.
   - Cerebellum: ataxia, dysarthria.
2. Manifestations of slight meningeal irritation: *may be present*.
3. Manifestations of viral infection:
   - *General:* fever, headache, myalgia, ….. etc….
   - *Specific:* depending on the causative virus, **e.g.**
     - LMNL paralysis with fasciculations: Poliomyelitis.
     - Parotid swelling: Mumps.

INVESTIGATIONS

1. **MRI:** of the brain.
2. **EEG:** is helpful.

TREATMENT

- Antiviral agent: Acyclovir.

COMA

DEFINITION

- A state of profound ***UNCONSCIOUSNESS*** from which the patient cannot be aroused, even by powerful stimuli.

PYSIOLOGICAL ASPECTS

- The state of ***CONSCIOUSNESS*** normally depends on:

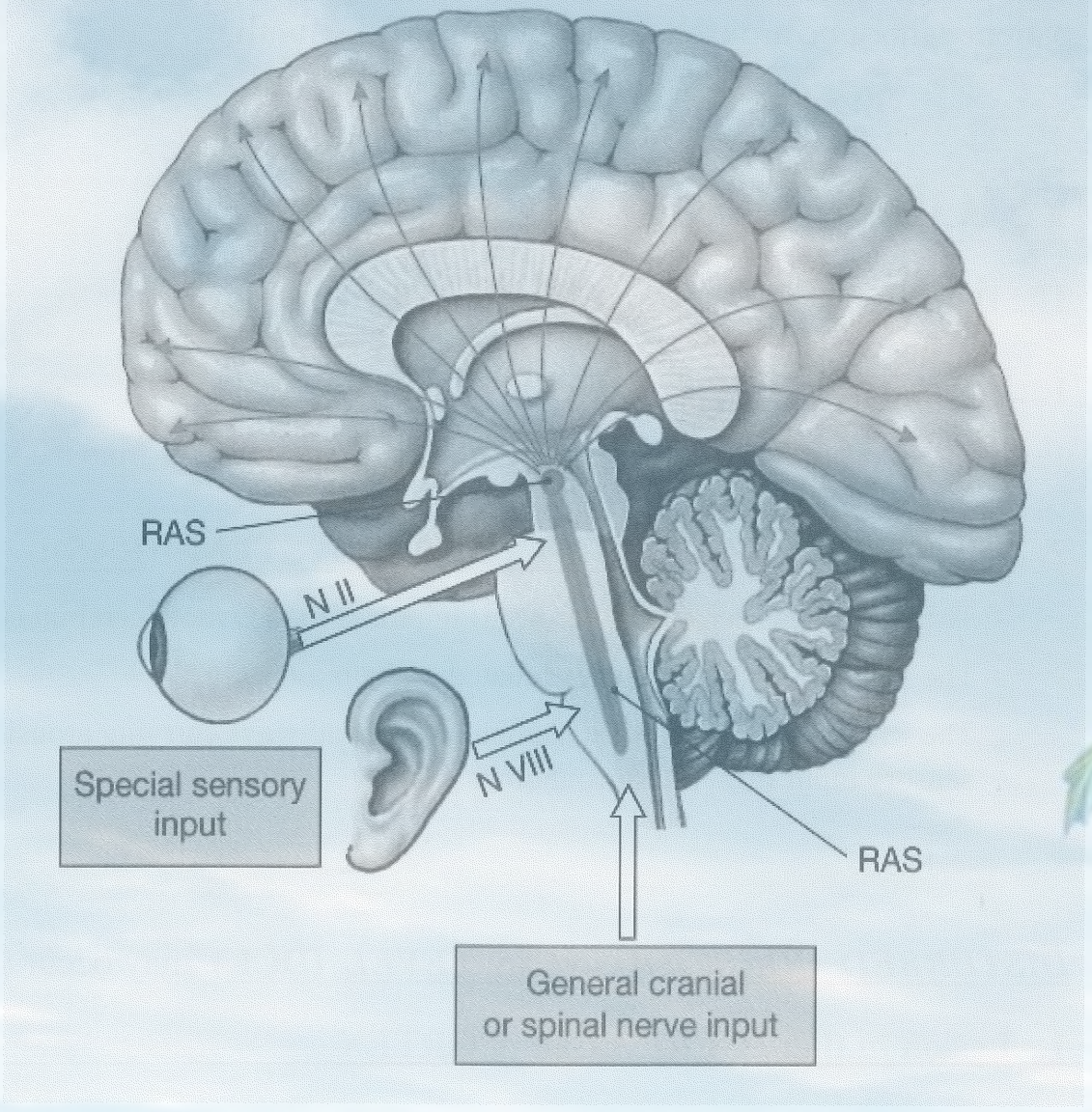
1. **Intact CEREBRAL CORTEX:**
   - The cerebral cortex receives activating (arousal) impulses from the RAS.
2. **Intact RETICULAR ACTIVATING SYSTEM (RAS):**
   - The RAS sends activating (arousal) impulses to the whole cerebral cortex.
   - The RAS is a group of nuclei situated in the brain stem.
   - The RAS receives stimulatory impulses via pathways carrying arousing sensory input from the surroundings.

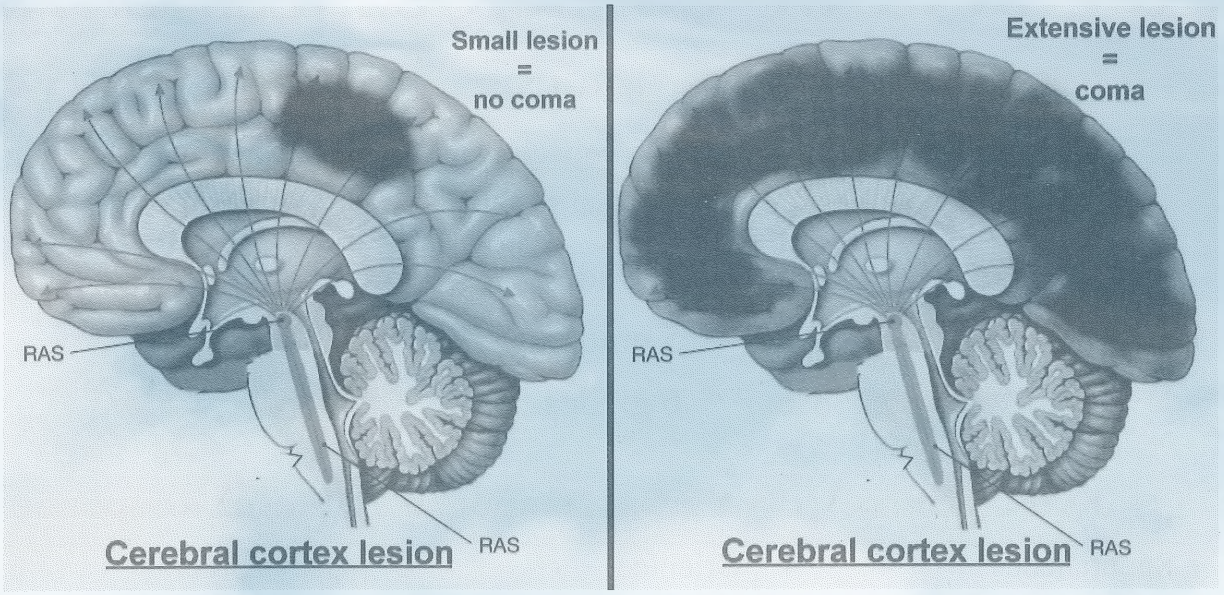
- ***DISTURBED CONSCIOUSNESS LEVEL*** (DCL) may occur due to:

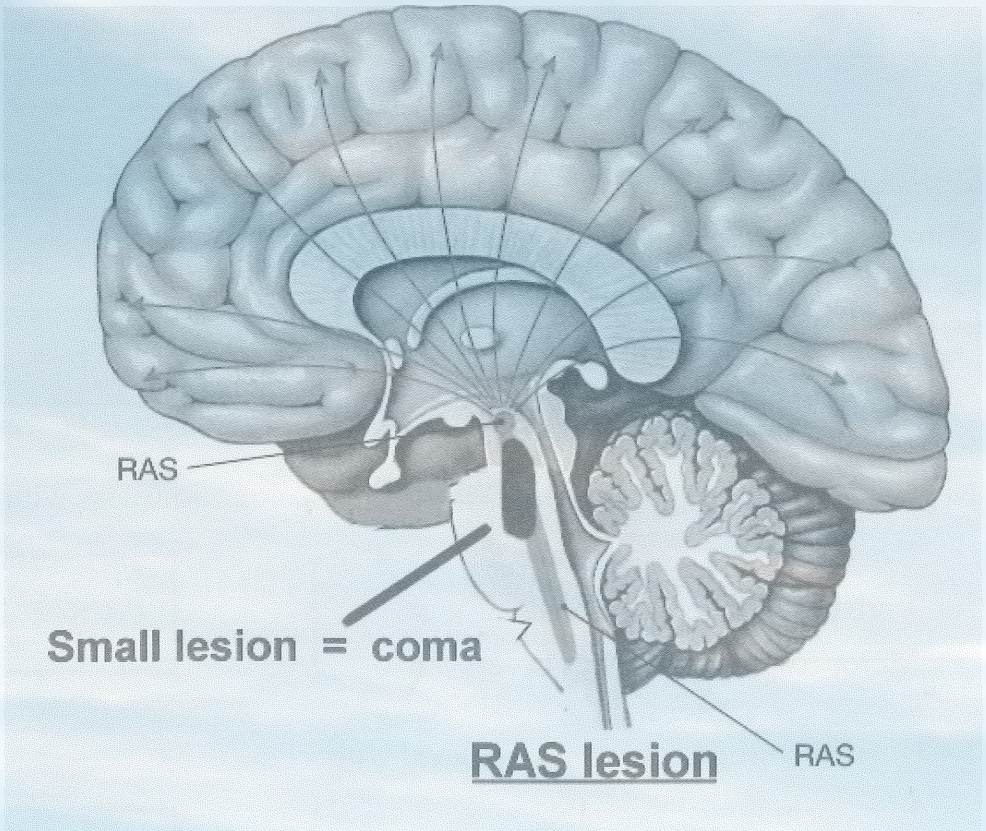
1. **Lesion in the: CEREBRAL CORTEX**
   - The lesion should be extensive to produce coma.
2. **Lesion in the: RETICULAR ACTIVATING SYSTEM (RAS)**
   - A small lesion is enough to produce coma.

STATE OF CONSCIOUSNESS

The RAS alerts the whole cortex




Small lesion
=
no coma
RAS
Cerebral cortex lesion
RAS
Extensive lesion
=
coma
RAS
Cerebral cortex lesion
RAS


RAS
Small lesion = coma
RAS lesion
RAS

GRADING OF CONSCIOUSNESS LEVEL

- **According to Glasgow coma scale (GCS):**

General grading of the level of **consciousness** is recently done according to the patient's response to external stimuli, using ***3 criteria:***

- Eye opening: 4 grades.
- Verbal response: 5 grades.
- Motor response: 6 grades.

Eye Opening		Verbal Response		Motor Response	
Spontaneous	4	Oriented	5	Obeys orders	6
In response to speech	3	Confused	4	Localizes to pain	5
				Withdraws to pain	4
In response to pain	2	Words, no sentences	3	Flexes to pain	3
		Sounds, no words	2	Extends to pain	2
None	**1**	**None**	**1**	**None**	**1**

- **According to Glasgow coma scale (GCS):**
  - Coma = complete loss of consciousness with:
    - Eye opening: none = 1
    - Verbal response: none = 1
    - Motor response: none = 1

    } **GCS = 3**

  - Normal consiousness (alertness) = complete consciousness with:
    - Eye opening: spontaneous = 4
    - Verbal response: oriented = 5
    - Motor response: obeys orders = 6

    } **GCS = 15**

  - DCL = disturbed consciousness level with:
    - Eye opening: variable
    - Verbal response: variable
    - Motor response: variable

    } **GCS = 4 – 14**

ETIOLOGY

Intracranial	Extracranial
Coma with lateralizing signs	*Coma without lateralizing signs*

1. INTRACRANIAL CAUSES

"Local causes in the brain"

- Traumatic: Head injury, e.g. *cerebral concussion, contusion, laceration.*
- Inflammatory: Encephalitis, meningitis, brain abscess.
- Neoplastic: Brain tumour.
- Vascular: Hemorrhage *(Cerebral & subarachnoid)*, thrombosis, embolism, Hypertensive encephalopathy.
- Epilepsy.

2. EXTRACRANIAL CAUSES

"General causes with 2ry effects on the brain"

A. Toxic:

1. Aspirin (salicylate) poisoning:
   - *Fever*, bleeding tendency, hyperventilation, vomiting.
2. Alcohol intoxication:
   - Characteristic mouth odour, flushed face & conjunctiva, ↑ blood alcohol.
3. Barbiturate poisoning:
   - Respiratory depression, circulatory failure, ↑ blood barbiturates.
4. Belladona (atropine) poisoning:
   - *Fever*, hot flushed dry skin, dilated pupils, tachycardia.
5. Carbon monoxide poisoning:
   - Cherry red skin.
6. Morphine (opiate) poisoning:
   - Pinpoint pupils, respiratory depression, hypothermia, bradycardia.

B. Metabolic: "METABOLIC ENCEPHALOPATHIES"

The term "metabolic encephalopathy" was elaborated by ***Kinnier Wilson*** * in 1912 to describe a clinical state due to various causes in which the brain's integrated activity is impaired in the absence of local structural abnormalities. Metabolic encephalopathy thus reflects diffuse (extensive) cerebral dysfunction induced by a systemic disturbance in metabolism.

1. Organ failure:

- Respiratory failure: *Hypercapnic encephalopathy.*
- Hepatic failure: *Hepatic encephalopathy.*
- Renal failure: *Uremic encephalopathy.*

2. Endocrinal:

- PITUITARY (Post): *Hypertonic encephalopathy in **DI**.*
- PITUITARY (Ant): *Panhypopituitarism.*
- THYROID: *Hypothyroidism & thyrotoxic crisis.*
- ADRENAL: *Addisonian crisis.*
- PANCREAS: *DIABETES* (**4 types of coma**).

3. Electrolyte & Acid – base disturbances:

- POTASSIUM: ↑ K, ↓ K.
- SODIUM: ↑ Na, ↓ Na.
- CALCIUM: *Hypercalcemic crisis.*
- Prolonged convulsions in TETANY → coma.
- Severe acidosis or alkalosis.

4. Other metabolic disturbances:

- Mitochondrial encephalopathy: a metabolic disorder caused by dysfunction of mitochondrial DNA.
- Wernicke's encephalopathy: due to *thiamine deficiency* or *alcoholism*.

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* **Samuel Alexander Kinnier Wilson** (1878 - 1937) was a British neurologist, he also described Wilson's disease.

C. Physical:

- Heat stroke.
- Hypothermia.

D. Cardiac auses:

- Cardiac arrest.
- Cardiac arrhythmias.

E. Infections:

- **M**eningitis.
- **M**alaria, especially the Cerebral type.
- **S**epticemia & fulminant infections.
- **S**tatus typhosus.

CAUSES OF FEBRILE COMA

A. Toxic:	Aspirin	&	Belladona poisoning.
B. Metabolic:	Hepatic failure	&	DKA.
C. Endocrinal:	Thyrotoxic crisis	&	Addisonian crisis.
D. Physical:	Heat stroke.		
E. Fevers:	ALL causes of fevers causing coma.		
F. Vascular:	Pontine hemorrhage	&	subarachnoid hemorrhage.

I. Any type of coma complicated by secondary infection, **e.g.** *hypostatic pneumonia*.

CLINICAL APPROACH TO COMA

INITIAL GENERAL MEASURES

- Ensure free airway passages.
- Ensure enough blood to the brain *(maintain adequate BP)*.

HISTORY TAKING

1. Onset:
   - *Sudden*: Cerebral hemorrhage
   - *Gradual*: brain tumour.
2. Head injury.
3. Convulsions: Epilepsy, brain tumours.
4. Drug intake: Drug poisoning.
5. Excessive exposure to the sun: Heat stroke.

GENERAL EXAMINATION

A) VITAL SIGNS

1. Temperature:
   - Hyperthermia: Causes of febrile coma.
   - Hypothermia: Hypothyroidism, Morphine poisoning.
2. Pulse:
   - Tachycardia: Thyrotoxic crisis.
   - Bradycardia: Hypothyroidism, Morphine poisoning.
3. Blood pressure:
   - High: Hypertensive encephalopathy.
   - Low: Addisonian crisis.
4. Respiration:
   - Deep, rapid: DKA, Renal failure.
   - Deep, slow: Barbiturate poisoning, Morphine poisoning.

B) MOUTH ODOUR

1. Acetone odour: DKA.
2. Alcohol odour: Alcohol intoxication.
3. Urineferous odour: Renal failure.
4. Fetor hepaticus: Hepatic failure.

C) SKIN EXAMINATION

1. Dry skin: DKA, Belladona poisoning.
2. Moist skin: Hypoglycemic coma.
3. Cherry red skin: Carbon monoxide poisoning.
4. Needle marks: Drug addiction.

D) EYE EXAMINATION

1. Pupils:
   - *Dilated fixed*:
     - Unilateral: 3rd nerve compression, as in uncal herniation.
     - Bilateral: Belladona poisoning.
   - *Constricted*:
     - Bilateral: Pontine hemorrhage & Morphine poisoning (pin point).
2. Fundus examination: papilloedema in cases of increased ICT.

CNS EXAMINATION

1. Signs of lateralization: denote an intracranial cause for the coma.

SIGNS OF LATERALIZATION

- EYES: Unequal pupils, Deviation of the eyes to one side.
- FACE: Facial asymmetry.
- MOTOR: Jacksonian fits, Unilateral paralysis or paresis.
- DEEP REFLEX & TONE: Asymmetry.
- SUPERFICIAL REFLEX: Unilateral positive Babinski

2. Signs of meningeal irritation: Meningitis, SAH.

INVESTIGATIONS

A) IMAGING

1. Plain x-ray skull:
   - Features of ICT: brain tumours.
   - Fractures: trauma.

2. MRI & CT scan: ***"The most important investigations"***
   - They are very accurate in diagnosing intracranial lesions.

B) LABORATORY INVESTIGATIONS

1. Blood examination:
   - CBC: leucocytosis in bacterial infections.
   - Blood film: malaria.
   - Blood levels of: glucose, urea & creatinine, bilirubin.
   - Blood gases: respiratory failure.
   - Toxicological studies: drug poisoning.

2. Urine examination:
   - Glucose.
   - Acetone.
   - Albumin.

3. CSF examination:
   - Bloody: SAH.
   - Purulent: Meningitis.

TREATMENT

1. Care for the comatosed (as in hemiplegia).

2. Treatment of the cause.

رقم الإيداع : ٢٠٠٩/٧٠٥٣

AL TASNEEM
PRESS

تليفون: ١٣ ٥٨ ٤٤ ٢٢ - ١٢ ٥٨ ٤٤ ٢٢